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REDUCTION OF PAIN AND OTHER UNDESIRABLE REACTIONS DUE TO PNEUMOENCEPHALOGRAPHY 755
Theodore J. C. von Storch and Howard H. Karr

THE ESTIMATION OF OPERATIVE RISK IN PATIENTS WITH CANCER 759
Allen S. Johnson and Herbert L. Lombard

THE PROTHROMBIN LEVEL IN EARLY INFANCY 762
Geoffrey Edsall

MEDICAL PROGRESS: NEUROSURGERY 766
Donald Munro

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL 771

EDITORIALS
Annual Meeting 777
Sale of Alcohol in Massachusetts 777

DEPARTMENTS
Obituary 778
Medical Eponym 780
Massachusetts Medical Society 781
Miscellany 786
Correspondence 786
Reports of Meetings 788
Notices 790
Book Reviews 791

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REDUCTION OF PAIN AND OTHER UNDESIRABLE REACTIONS DUE TO PNEUMOENCEPHALOGRAPHY*

THEODORE J. C. VON STORCH, M.D.,† AND HOWARD H. KARR, M.D.‡

BOSTON

SINCE its introduction by Dandy[1] twenty years ago, pneumoencephalography has become an accepted diagnostic procedure in the practice of neurology, neurosurgery and psychiatry. When properly performed, it results in a mortality of less than 0.3 per cent.[2–4] The indications for encephalography are numerous, the contraindications few.[2, 3, 5–11] Unfortunately, encephalography is an extremely painful procedure. When pain no longer functions as a defense mechanism, it becomes a pathologic process, the existence of which is unjustifiable. Furthermore, continued pain during encephalography causes marked fluctuations in respiration and intravascular pressure, and is, perhaps, productive of shock. Other accessory reactions to encephalography are hyperthermia, nausea, vomiting, aseptic meningitis and, occasionally, urinary retention and shock. Physicians who fail to follow the postencephalographic course of their patients are too often unimpressed by the necessity of reducing the subsequent reactions. Others are overly impressed and hesitate to utilize this valuable diagnostic method. Various means have been used to diminish the undesirable reactions of encephalography. It is our purpose to analyze these methods and to draw therefrom certain conclusions, which eight years' experience has shown to be of practical value.

CONTRAINDICATIONS AND PRECAUTIONS

Tumor of the posterior fossa and evidence of marked increased intracranial pressure are contraindications to encephalography. Patients with advanced hydrocephalus do not do well, as a rule. In such cases, especially in children, it is necessary to force fluids after the procedure, to replace the relatively large volume of fluid that has been lost. Difficulties may be encountered when marked cerebral arteriosclerosis is present. Hemorrhage may be met with in marked arterial hypertension or in thin-walled intracranial aneurysms. In general, cases with brain tumor react more severely than those without tumor. Nevertheless, when proper precautions are taken, encephalography may be safely performed in any of these conditions except tumor of the posterior fossa or marked increase in intracranial pressure.

PRE-ENCEPHALOGRAPHIC CONSIDERATIONS

When apprehension is a significant factor it may be diminished by the oral administration of from 1½ to 3 gr. of Nembutal (sodium ethylmethylbutylbarbiturate, Sodium Amytal (sodium isoamylethylbarbiturate) or another suitable sedative on the night before encephalography.[2, 6, 12, 13] A repetition of the same dose of sedative about two hours before the procedure is occasionally necessary. To avoid the untoward results of vomiting, the previous meal should be omitted and fluids limited. Vomiting can be prevented in most cases by the subcutaneous injection of from 1/150 to 1/50 gr. of atropine sulfate, one hour before lumbar puncture. The use of the larger dose, in adults, diminishes any tendency toward laryngeal spasm, which may occur when the nonvolatile intravenous anesthetics are employed. Atropin also decreases nasopharyngeal and oral secretions, which may complicate the use of any anesthetic.

ENCEPHALOGRAPHIC CONSIDERATIONS

In co-operative subjects, lumbar puncture after local novocain infiltration is usually less difficult than in relaxed anesthetized subjects. With uncooperative subjects it is advisable to induce anesthesia before attempting the puncture.

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Analgesia

Morphine,[2, 14] scopolamine,[11, 13, 15, 16] chloral hydrate,[8, 9, 12] oral Nembutal,[6] and Amytal[4, 7, 17] have been recommended as analgesic agents. Partial analgesia or narcosis is easier to obtain than complete anesthesia, and the patient requires less attention. On the other hand, it produces an incompletely relaxed patient, who is not infrequently unco-operative. The patient still suffers unnecessary pain and often requires further analgesia or the use of aromatic spirits of ammonia.[2, 8] Epileptic and psychotic patients often become unmanageable under barbiturates. Morphine causes an additional respiratory strain, and is indicated only when barbiturates cannot be used. Therefore, it is our opinion that general anesthesia is preferable to partial analgesia.

Anesthesia

General anesthesia entirely relieves the pain of encephalography, contributes largely to its prevention in the postencephalographic period, prevents undue haste due to the patient's repeated complaints, and prevents struggling that tends to cause intracranial hemorrhage or to dislodge the drainage needles. On the other hand, accidents may occur if the operator is unfamiliar with the use of the anesthetic employed. Some type of support is necessary because of the relaxation of the patient — several such supports have been described.[12, 18–21] The constant attention of the anesthetist is necessary for the proper induction and maintenance of the anesthesia, especially when the intravenous route is used. Nevertheless, the advantages may be said to outweigh the disadvantages. However, the choice of anesthetic is of great importance in obtaining satisfactory results.

Inhalation anesthesia. Ether,[3, 22–25] ethyl chloride[26] and nitrous oxide[27] have been recommended for inhalation anesthesia. All these gases increase arterial, venous and cerebrospinal-fluid pressures to a marked degree and in a fluctuant fashion. These increases interfere with any simultaneous-replacement method, prevent the use of automatic methods[13] and predispose to intracranial hemorrhage, cerebral edema and herniation of the medulla. Vomiting is not infrequent, and respiratory complications may occur. A constant level of anesthesia is difficult to maintain, and the postencephalographic period is apt to be stormy. For these reasons, it is advisable to use inhalation anesthetics in encephalography only as a brief induction to other analgesic methods.[22]

Rectal anesthesia. Avertin (tribromethanol in amylene hydrate) has been recommended for this purpose.[25, 28, 29] After instillation, the operator has very little control over the depth of anesthesia. Therefore, the tendency is to give too little, with the result that the patient emerges before completion of encephalography, making it necessary to use supplementary inhalation anesthesia. Emergence usually occurs just as roentgenography is attempted, with the result that the films are spoiled by movement of the patient. Furthermore, because the patient is in the sitting position, the anesthetic fluid is often expelled during the encephalographic procedure. For these reasons, and others enumerated by Sise,[30] most clinics have justifiably abandoned the use of rectal anesthesia for encephalography, except in children in whom intravenous anesthesia may be impractical or dangerous.

Intravenous anesthesia. This method has been used, in recent years, with increasing frequency. Sodium Amytal,[14, 28] Nembutal,[13, 28, 29] Evipal Sodium (sodium methylcyclohexenylmethylbarbiturate)[29, 31, 32] and paraldehyde[33, 34] have been used. In the last few years, several clinics have used Pentothal Sodium (sodium ethylmethylbutylthiobarbiturate) extensively. Our experience with the last drug has led us to believe that, with proper precautions, it is the best available anesthetic for pneumoencephalography, and we are in complete accord with Nicholson and Sise,[32] who have recently reported excellent results in 177 cases. Contraindications to its use include any condition that may encroach on the airways, gross hepatic disease, severe anemia, severe renal disease, extreme debility and cardiac decompensation. These, however, are contraindications to encephalography itself. Although not recommended in children under seven years of age, it has been used successfully in an occasional case. Rectal Avertin is preferable in patients under seven. In persons with low blood pressure, the introduction of gas into the cranium is usually sufficient to re-elevate the pressure. Respiratory depression or laryngeal spasm occasionally occurs. Preoperative administration of from 1/150 to 1/50 gr. of atropine sulfate is effective in preventing laryngeal spasm. Infrequently, it is necessary to keep the airways open by catheterization. Ten per cent carbon dioxide with oxygen should be kept at hand but rarely needs to be utilized. One or two cubic centimeters of 25 per cent Coramine (pyridine-beta-carboxylic acid diethylamide) intravenously will rapidly counteract the effects of Pentothal Sodium, if necessary. In our experience the most important factor in the use of Pentothal Sodium is the maintenance of a slow, almost continuous, intravenous administration. Untoward results are rare, and are almost invariably due to rapid injection. A dangerous dose of these

drugs is dependent not so much on the total amount as on the rate of injection.[30]

The following method is recommended:

From one to one and a half hours after administration of 1/150 to 1/50 gr. of atropine sulfate, the patient is placed erect in the chair for encephalography. Several arterial pressure determinations are made, the elbow is splinted in extension and venipuncture is performed at the brachial vein. A 5 per cent solution of Pentothal Sodium in sterile normal saline solution is then slowly introduced by almost imperceptible movement of the plunger of a 20-cc. syringe. The rate is maintained or lessened according to the depth of anesthesia. Injection is continued during the replacement of cerebrospinal fluid, and the needle withdrawn just before roentgenography. Observations are occasionally made of the arterial tension, and the mouth and neck are watched to prevent airway obstruction.

This method results in a pleasant induction and a uniform maintenance of a relaxed anesthetic state. Complete relaxation is obtained during the subsequent roentgenography, which immediately follows replacement of the cerebrospinal fluid.[13] No subsequent analgesia is needed for from four to twelve hours after the procedure. The cerebrospinal-fluid pressure is unaltered, and the arterial tension is apparently unaffected. Respiratory depression has been rare in our experience. Therefore, although the use of Evipal or paraldehyde deserves further consideration, at present Pentothal Sodium seems to be the most effective means of obtaining adequate anesthesia during pneumoencephalography.

Type of Gas Used

Filtered and unfiltered air, carbon dioxide, nitrogen and helium have been used to replace the cerebrospinal fluid.[35] Ethyl chloride, divinyl chloride, vinyl chloride, acetylene, cyclopropane and ether have been used in animals,[36] nitrous oxide and ethylene in human beings.[22] Freon (dichlorodifluoromethane) has been used by one of us.[37] Many clinics use oxygen, but most continue to utilize room air. Of these gases, unfiltered room air is used most frequently but causes the severest reactions; filtered air causes some diminution of the reaction; helium and oxygen are said to be less irritating than air; nitrous oxide is better than oxygen, and according to the results in over 600 cases, ethylene is least irritant.[22] The remaining gases are either too dangerous, or not sufficiently better than air, to warrant consideration.

Aird[22] has used pure ethylene to replace the cerebrospinal fluid in over 600 cases, with excellent roentgenographic results and a marked decrease in the accessory effects of encephalography. Although ethylene is highly explosive when mixed with air, it is not explosive when maintained in the pure form in a closed system. There have been no untoward results as yet from the use of this gas. The use of such an anesthetic gas does not, however, render anesthesia (Avertin fluid) unnecessary, although in children an induction anesthesia of ether is sufficient. The real value of ethylene is in reduction of postencephalographic reactions. Because of the fear of explosion, very few workers have used this method. Aird's results suggest that such fear may be unfounded.

It may be said that although, because of inertia and convenience, air continues to be the gas most frequently used in encephalography, other gases lessen the reactions afterward and diminish them somewhat during the procedure. Oxygen is preferable to air from this point of view, and if the fear of explosion can be overcome, ethylene is perhaps the gas of choice.*

Amount of Gas Used

The reaction to encephalography varies directly with the amount of gas used.[2, 38] The use of small volumes in the so-called "ventricular repéráge" results in a minimum of discomfort, some patients being up and about within several hours after injection.[39] On the other hand, injection of small amounts of air is in most cases inadequate for the diagnosis of more than the most obvious intracranial masses or gross ventricular enlargement.[13] After the introduction of 100 cc. of gas, additional replacement causes little change in the patient's immediate reactions. Additional gas does, however, prolong the subsequent discomfort. It may be said, therefore, that even though less gas diminishes the subject's reactions, it is necessary in all but a few cases to replace completely the cerebrospinal fluid with whatever amount of gas may be necessary.

Method of Cerebrospinal-Fluid Replacement

Numerous observations have demonstrated that the two-needle, simultaneous method of replacing cerebrospinal fluid by gas diminishes the patient's reactions during and subsequent to encephalography.[13, 38] Several types of apparatus have been described that render replacement more or less automatic.[13, 40–43] To avoid mechanical irritation of the brain, which, after replacement of cerebrospinal fluid, is lying in a "dry box," and to avoid undue tension on the meninges and on the vessels at the vertex, a minimum of manipulation should be maintained. Slow, continuous anteroflexion and posteroflexion of the head is sufficient for adequate ventricular replacement. Transportation

*It should be emphasized that when readily absorbable gases are used, such as oxygen or to a greater degree ethylene, the roentgenologic technic must be accelerated accordingly.

and handling of the patient should be at a minimum.

Postencephalographic Considerations

As has been noted, anesthesia with intravenous Pentothal Sodium diminishes the sequelae of encephalography. The use of ethylene to replace the cerebrospinal fluid exerts most of its effect in diminishing postencephalographic reactions. Avoidance of unnecessary cranial manipulation tends to decrease subsequent ill effects, and the use of the simultanous-replacement method does likewise.

If shock occurs, the use of Coramine, caffeine sodium benzoate and warmth is usually sufficient therapy. It has been found unnecessary to elevate the foot of the bed. Only in rare cases has it been necessary to administer clysis to adults. In children, fluids are sometimes useful. In children with hydrocephalus, it is essential to administer fluids at once because of the dehydration that occurs when the body attempts to restore the relatively immense volume of cerebrospinal fluid that has been replaced by gas. In adults with hydrocephalus, fluids are helpful but not essential. Lumbar puncture after encephalography usually reveals a lowered rather than an increased intracranial pressure and is therefore of no value except in rare cases of increased pressure due to subsequent edema or hemorrhage.[44] In every case it is advisable to observe the temperature, respiration, arterial pressure, pulse pressure and pulse rate carefully for twenty-four hours after encephalography. A wet icecap is helpful if applied immediately on return to the ward. Codeine and acetyl salicylates may be given when necessary. Three grains of Nembutal by mouth is helpful. Morphine is to be avoided.

If the patient is allowed to breathe 95 per cent oxygen for two or three hours after encephalography, the intracranial gas is rapidly removed, and the subsequent course is greatly improved.[45] The oxygen can be more readily administered through a Boothby respirator mask than by the apparatus described by Schwab, Fine and Mixter.[45] Seven to eight liters of 100 per cent oxygen are delivered per minute.

After all reaction to encephalography has disappeared, the patient may complain of a postlumbar-puncture headache that occurs only in the erect position. This can be relieved by the intravenous injection of 7½ gr. of caffeine sodium benzoate from one to three times a day or by the resumption of recumbency.

Summary

So that pneumoencephalography may be available for use in all cases in which it promises to be of value, every effort should be made to reduce the dangers, pain and other untoward reactions. This may be accomplished to a large degree by proper consideration of the contraindications to encephalography; the liberal use of preliminary sedation, for which Nembutal has been found most useful; prophylactic injection of from 1/150 to 1/50 gr. of atropine sulfate; the use of an effective intravenous anesthetic, such as Pentothal Sodium; the use of a suitable gas, such as air, oxygen or possibly ethylene; simultaneous replacement of the cerebrospinal fluid by gas; avoidance of undue manipulation of the patient; careful postencephalographic observation and the use of analgesics and sedatives, when necessary; the use of fluids and shock therapy, when indicated; and the inhalation of 95 per cent oxygen, when necessary.

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THE ESTIMATION OF OPERATIVE RISK IN PATIENTS WITH CANCER*

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THE operative mortality in a hospital limited to the treatment of cancer is necessarily high, but a preliminary review of a few hundred cases gave the impression that a lower mortality could be obtained by limiting operations to selected patients without obesity, malnutrition, hypertension or a history suggesting cardiac disease. This study was undertaken to prove whether or not this thesis was correct. The material consisted of 2445 cases subjected to major operations at the Massachusetts state cancer hospitals at Pondville (1927 to 1939) and at Westfield (1937 to 1939).

Operative mortality is a term lacking in precise definition. In this study we have used the criteria of Warren,[1] who considered that patients dying within one month after operation were operative deaths, provided the death was not due to the natural course of the condition for which the operation was done. According to this definition, a patient dying of pneumonia or heart failure, obviously precipitated by the operation, is listed as an operative death. This is true of sepsis having a causal relation to the operative procedure. It has also seemed justifiable to list as an operative death an occasional patient who survived the arbitrary one month, but who pursued a progressively downhill course with one postoperative complication after another and finally died of the complications and not of the original disease that necessitated the operation. Because a patient who has developed fatal lung abscesses after resection of the rectum is still breathing, although moribund, thirty days later, it does not seem reasonable to absolve the operation from responsibility for the death. On the other hand, patients were not so listed who, after a palliative operation, died of sepsis due to the original lesion and antedating the surgical procedure.

Although a variety of fatal lesions were often present at the time of death, some were listed by the pathologist as the "immediate cause of death" in the autopsied cases, which comprised 58.5 per cent of the fatalities. In the cases not subjected to autopsy it was necessary to rely on a careful appraisal of the data in the histories. For example, a patient with obvious sepsis was not listed as dying of heart failure just because his heart finally stopped beating. On the other hand, the classic clinical picture of congestive heart failure, although it might have been precipitated by postoperative sepsis, was listed as the cause of death if the patient succumbed under these circumstances. In the hope of eliminating inconsistencies resulting from appraisal by different people, one of us (A. S. J.) undertook the entire task of evaluating the clinical data, and the other (H. L. L.) assumed the responsibility for the statistical computations.

TABLE 1. *Operative Mortality Subdivided by the Age of the Patient.*

AGE	NO. OF CASES	NO. OF DEATHS	PER CENT MORTALITY
Under 40 years	270	11	4.1
40–49	467	33	7.1
50–59	626	84	13.4
60–69	713	173	24.3
70 and over	368	85	23.1
Totals	2444*	386	

*In 1 case the age was unknown.

It is well recognized that increasing age produces an increase in surgical mortality. Table 1

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shows the operative mortality according to age groups. Mortality increases progressively up to the age of sixty; from then on it is practically stationary.

A history of cardiac disability has been considered by many observers to increase operative risk. In this group were listed all patients who gave a history of exertion dyspnea, cyanosis, angina or edema that could be ascribed to heart disease. Dyspnea or cyanosis due obviously to a pulmonary lesion or edema due to hypoproteinemia or varicose veins was naturally excluded. The patient's history was given more consideration than the physical or laboratory examinations for two reasons. First, as Marvin[2] has emphasized, the history is more important than the physical examination in determining the patient's ability to withstand anesthesia and operation; and Sprague[3] has pointed out that "the ability of the patient to carry on his daily activity without symptoms usually tells more about his myocardial power than can be discovered on examination of the heart." Secondly, it will be some time before electrocardiographic, x-ray and other laboratory procedures are available in every case subjected to major surgery.

A patient was not considered hypertensive unless a systolic pressure of 160 or more was accompanied by a diastolic pressure of 100 or more. These levels were deliberately made higher than life-insurance norms in an attempt to exclude transient elevations due to emotional disturbances. Furthermore, if only one or two readings were above this level and the majority were lower, the patient was not classed as hypertensive.

The classification of the patient's nutritional state may not receive general acceptance. Lack of data made it impossible to classify each patient in terms of percentage above or below norms for a given age, height and sex. They were therefore listed according to the examining physician's appraisal, which was either "obese," "well-developed and well-nourished" or "poorly nourished." That this inexact classification of obese or malnourished implied a significant departure from normal may be inferred from the fact that this required a detailed note in longhand in contrast to the check marks that were used on a printed form when the patient appeared normally nourished.

The duration of the operation was likewise found to influence surgical mortality, probably because duration is likely to parallel the severity of the procedure.

In Table 2 the operative mortality for all cases is contrasted with that for the various groups. The mortality for long operations (two hours or more) was very high (46.7 per cent), in contrast to 12.1 per cent for all operations under two hours; and those patients with a cardiac history, malnutrition or an age of sixty or over had a considerably higher mortality rate than did the total population of the study. The mortality of the obese group was below that for the total population, and the rate of the hypertensive group was practically the same as that of the total.

TABLE 2. *Operative Mortality.*

CLASSIFICATION	NO. OF CASES	MORTALITY %
Total cases	2445	15.8
Obesity	266	7.9
Malnutrition	565	25.3
Old age (over 60)	1081	23.8
Hypertension	347	16.4
Cardiac history	346	24.6
Long operation (over 2 hours)	244	46.7
Short operation (under 2 hours)	2201	12.1

With the realization that combinations of these variables were common and that the true picture might easily be masked because of the interrelation between them, the variables were subjected to partial association. For example, a group of patients might all have cardiac disease and hypertension and be aged. The problem was to determine whether all three variables contributed to the high operative mortality of the group, or whether only one was responsible, the seeming relation of the other two being simply due to the combined presence of all three variables. It was recognized, moreover, that differences in mortality inherent in the type of operation must be taken into account in these calculations. The analysis of plural variables, in which the data are grouped as present or absent, requires working with partial association, which is the effort to eliminate the fallacy of mixed classification by using partial universes.

Subuniverses were constructed, and the mean of the combination of all the universes for a given variable was compared with its standard deviation. The criterion of measurement of significance was 2.6, p equaling 0.01. The results follow: obesity −0.8; malnutrition 1.97; old age (over sixty years) 3.5; hypertension 1.17; cardiac history 1.86; long operation (over two hours) 4.0. Only the figures for the aged and for length of operation are significant. The clinician who may have been frightened off by the statistical method of approach is referred to Table 3 in which the operative mortality is given for each pure subgroup. All patients in any given group in this table were afflicted with only one of the presumable contraindications to operation. The conclusions to be drawn from

contrasting these mortality rates with the total mortality of 15.8 per cent are substantially the same as those already reached by the method of partial association.

This clearly indicates that the only variables of importance in this selection of cases were the prolonged operation and the age of the patient. It is impossible to postulate from this study whether similar results would be obtained in another series, since the choice of patients might easily vary.

Table 4 lists the immediate causes of death in their order of importance for the total 386 deaths.

TABLE 3. *Relation between Operative Mortality and "Pure" Presumable Contraindications to Surgery.*

CONTRAINDICATION	NO. OF CASES	MORTALITY
		%
Obesity alone	113	2.7
Malnutrition alone	210	14.8
Old age (over 60 years) alone	429	21.7
Hypertension alone	80	11.2
Cardiac history alone	59	15.3
Long operation (over 2 hours) alone	62	27.4

TABLE 4. *Causes of Postoperative Deaths.*

CAUSE OF DEATH	TOTAL CASES (386)	AUTOPSIED CASES (226)
	%	%
Sepsis	35.8	40.3
Pneumonia	26.9	26.1
Cardiac failure	11.1	11.1
Pulmonary embolism	7.3	7.5
Hemorrhage	4.9	5.7
Renal failure	3.1	3.1
Surgical shock	4.9	4.9
All other causes	6.0	1.3

Warren[1] in a study of 252 postoperative deaths coming to autopsy found the immediate cause of death to be in order of their incidence: sepsis, pulmonary embolism, pneumonia and cardiac failure.

Table 5 shows the distribution of these causes among our patients having to their discredit only malnutrition, hypertension, cardiac history, old age, long duration of operation, or obesity. As might be expected, the same approximate relation exists for the chief causes of death: sepsis, pneumonia, heart failure and pulmonary embolism. The low incidence of heart failure among the "pure" cardiac patients and "pure" hypertensive patients coincides with the observations of Butler, Feeney and Levine[4] that, even among patients with heart disease, heart failure was seldom the cause of postoperative death. Brumm and Willius[5] also found only 4.5 per cent cardiac deaths postoperatively among 257 patients with severe coronary disease. These observations must be contrasted with Sprague's[3] report of heart failure's responsibility for 50 per cent of the postoperative deaths in a group of 170 cardiac patients.

The mortality for different types of anesthesia is listed in Table 6. One must be cautious about drawing conclusions from these figures, since so-called "safe" anesthetics like novocain were often used in emergency or palliative operations when the patient was in a very poor condition. The strikingly higher mortality in cases in which more than one anesthetic was employed was probably due to difficulties inherent in the procedure, which increased the length of the operation so that spinal anesthesia had to be supplemented with general or with local anesthesia. The importance of the duration of the operation has already been discussed. Because a given operation usually postulated a certain type of anesthesia, it was not possible to compare the hazard of different types of anesthesia in a particular operation in which the expected mortality was well established.

TABLE 6. *Mortality with Different Types of Anesthesia.*

ANESTHESIA	NO. OF CASES	NO. OF DEATHS	MORTALITY
			%
General	1339	148	11.1
Local	524	102	19.5
Spinal	339	75	22.1
Mixed (more than one)	227	58	25.6
Totals	2429*	383	

*In 16 cases there was no record of the anesthesia.

TABLE 5. *Causes of Postoperative Death Subdivided According to Criteria of Operability.*

	TOTAL DEATHS	CAUSES OF DEATH				
		SEPSIS	PNEUMONIA	HEART FAILURE	PULMONARY EMBOLISM	ALL OTHERS
Obesity alone	3	1	–	–	1	1
Malnutrition alone	31	12	10	–	2	7
Old age (over 60 years) alone	93	37	26	10	8	12
Hypertension alone	9	5	–	1	1	2
Cardiac history alone	9	4	2	–	–	3
Long operation (over 2 hours) alone	17	7	6	–	–	4

Since the age of the patient and the duration of the operation appear to be the most important factors in determining operative mortality, the interrelation of these factors demands examination. With an operation that lasted longer than two hours, the mortality among patients over sixty

years of age was 1.7 times as great as that among those under sixty. Among those with an operation of less than two hours' duration, the mortality rate of patients over sixty was 2.6 times as great as that of those under sixty. A comparison of the over-sixty group with over two hours' operations with the over-sixty group with less than two hours' operations shows the former to be 2.9 times as great. It appears that both variables are of great significance in operative mortality, and the long operation is probably the more important of the two.

Summary and Conclusions

In the work done at the Pondville and Westfield cancer hospitals, such variables as obesity, malnutrition, cardiac history and hypertension are apparently of little value in determining operative mortality; the age of the patient and the length of the operation are of considerable importance, with the latter perhaps the more significant of the two.

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THE PROTHROMBIN LEVEL IN EARLY INFANCY*

Its Relation to Hemorrhage and Other Neonatal Disturbances

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Whipple[1] observed over twenty-five years ago that hemorrhagic disease of the newborn might be associated with an extremely low plasma prothrombin. Brinkhous et al.[2] using a method[3] of prothrombin titration much more precise than any previously employed, demonstrated that the majority of newborn infants had moderately low prothrombin titers. Moreover they found that one infant, suffering from hemorrhage, had only 5 per cent of the normal adult level. The present report is the result of an attempt to define further the relation between neonatal hemorrhage and plasma-prothrombin deficiency.

Material and Methods

Prothrombin determinations were made on bloods obtained from 27 infants at the Infants' Hospital, the Children's Hospital and the Boston Lying-in Hospital.‡ Blood drawn from the internal or external jugular vein was added to about one tenth of its volume of 3 per cent sodium citrate in a graduated tube, and centrifuged for twenty to thirty minutes at 2000 revolutions per minute; the hematocrit was noted, and the plasma drawn off. The method of prothrombin titration was modified slightly from that of Quick.[4] Plasma in 0.5-cc. amounts was placed in clean test tubes in a water bath at 37°C. To each 0.5 cc. of plasma was added enough 0.9 per cent sodium chloride solution to give a final volume of 1 cc. after the further addition of 0.2 cc. of human placental coagulant[5] and approximately 0.05 cc. of 2.5 per cent calcium chloride solution. The prothrombin time was taken as the interval between the addition of calcium chloride and the gelation of the contents of the tube. The optimum amount of calcium chloride to be added was determined for each plasma, since it was found, in agreement with others,[6, 7] that unless this precaution was observed distorted prothrombin times might be obtained. Human placental extract (freshly prepared each week) was employed rather than animal tissue, since it gave a longer prothrombin time, which could be measured with greater accuracy. Citrate was preferred to oxalate, since it forms no precipitate on the addition of calcium ions.

The empirical correlation between prothrombin time and plasma-prothrombin concentration was determined by testing serial dilutions of normal plasma with prothrombin-free plasma. Plasma was rendered prothrombin free by absorption with alumina[8] or by repeated passage through a Seitz filter.[9] The values obtained in several such test runs are presented in Table 1, in the form of the ratio of the clotting time of the sample with diminished prothrombin to that of the normal control. The values presented differ somewhat from those described in the earlier reports on the subject[4, 10]

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but correspond closely to those published by Quick[11] in 1938.

Estimates of plasma prothrombin were reported in terms of the percentage of the adult normal, to the nearest 5 per cent, except that values from 70 to 100 per cent were entered as "70+," accurate determinations in these ranges being unreliable unless a more refined method is employed. It was found impossible to establish with certainty a critical level of plasma prothrombin, below which bleeding might be regularly expected; in various reports on this subject the "critical level" is given as 20 per cent,[12, 13] 35 per cent,[14] 30 to 50 per cent[15] and 50 per cent.[16] In the absence of any general agreement on this point, the level of 30 per cent was tentatively selected as being most consistent with the available findings.

TABLE 1. *Prothrombin Concentration and Prothrombin Time: Experimental values obtained with mixtures of normal plasma and prothrombin-free plasma, expressed as the ratio of the prothrombin time obtained with diminished prothrombin concentration to that obtained with 100 per cent prothrombin concentration.*

Prothrombin Concentration %	Test I* 5-5-38	Test II† 7-26-39	Test III‡ 7-26-39	Test IV† 9-28-39
90	1.04:1	—	—	—
80	1.06:1	1.00:1	—	1.09:1
70	1.10:1	—	—	—
60	1.18:1	1.21:1	1.26:1	1.24:1
50	1.28:1	—	—	—
40	1.45:1	1.53:1	1.52:1	1.50:1
30	1.65:1	—	—	—
20	1.95:1	2.05:1	1.96:1	1.94:1
10	3.00:1	—	—	—

*Cow plasma, clotted with bovine brain extract.
†Human plasma, clotted with placental extract.
‡Same plasma as in Test II, clotted with an old, partly deteriorated preparation of placental extract. Note that deterioration of placental extract did not significantly alter the ratio of prothrombin time to prothrombin concentration.

Results

Thirty-nine blood samples from 27 infants less than five weeks old were titrated; the data are presented in Table 2. All specimens were obtained from infants under special observation, and hence the data do not represent a cross section of normal infancy in this age group. Neither infants nor mothers received vitamin K during this study. In Cases 1 to 6 the patients were apparently healthy infants. In Case 7 the child had a colon-bacillus septicemia from which he recovered. Cases 8 to 19 included 12 patients manifesting signs of hemorrhage other than intracranial bleeding alone.

Eleven infants (Cases 12, 13, 15, 16 and 21 to 27) were diagnosed as having some intracranial injury (neonatal asphyxia or intracranial bleeding). The patients in Cases 18 and 20 vomited all feedings from birth for about one week. In Case 19 the infant was first seen at the Infant's Hospital when nineteen days old. Early neonatal data could not be obtained, but a tentative diagnosis of erythroblastosis was made. Dam, Tage-Hansen and Plum[17] have recently published the finding of a very low plasma-prothrombin titer in this condition.

The extreme and average prothrombin levels, average birth weights and duration of labor for each of the above groups are summarized in Table 3.

TABLE 3. *Correlation of Condition of Infants with Plasma-Prothrombin Levels, Average Birth Weight and Average Duration of Labor.*

Condition of Infant	No. of Cases	Prothrombin Levels: Extremes %	Prothrombin Levels: Average %	Average Birth Weight lb.	oz.	Average Duration of Labor hr.	min.
Apparently well	6	20–70+	49	7	5	8	10
Hemorrhage	12	5–70+	27	8		13	50
Intracranial injury	11	5–70+	29	7	14	13	35
Vomiting	2	5–15	—	8	9	—	

Discussion

The above data indicate a wide variation in the plasma-prothrombin level in early infancy, both in normal and in abnormal infants. Quick[18, 19] has reported a high prothrombin level at birth, followed by a drop in the next forty-eight to seventy-two hours, and a subsequent rise toward the normal adult level during the ensuing week. These findings show a close correlation with the known variation in bleeding and clotting times during the neonatal period.[20, 21]* In the cases here presented, however, such a relation was not regularly observed. Whereas in Cases 2, 15, 16 and 23 a low prothrombin in the first two or three days was succeeded in two to five days by a considerably higher level, Cases 12 and 20 exhibited a stationary or even a falling titer over a similar period. Thus, although Quick's observations probably represent the normal course of prothrombin fluctuation in young infants, this sequence of events may be disrupted by pathologic disturbances in early neonatal life. This possibility is borne out further by the prothrombin levels observed in the 11 patients showing evidence of intracranial injury. As seen in Table 3, the average prothrombin titer in this group was abnormally low. This observation may be correlated with the recent substantiation by Clifford[23] of the observation that a large pro-

*Ziffren et al.[18, 22] find neonatal prothrombin titers by their method to be much lower than those reported by various workers using Quick's method. They question the validity of the latter method for prothrombin determinations on the newborn, and offer evidence that the low neonatal prothrombin titer may be in effect partially offset by an increased "convertibility" of the prothrombin at birth. Pending further data on the validity of the various methods for prothrombin estimation, the use of Quick's method seems satisfactory, particularly since it represents an actual measure of the efficacy of the prothrombin.

TABLE 2. *Clinical and Laboratory Data on Cases Studied.*

Case No.	Complications of Pregnancy	Delivery	Duration of Labor	Birth Weight	Clinical Condition	Age of Infant	Prothrombin Level
			hr.	*lb. oz.*		*days*	%
1	Latent syphilis	Forceps	11	7 4	Normal	⅛ 3	70+ 70+
2	Latent syphilis, toxemia	Normal	2	6 10	Normal	½ 4	20 35
3	None	Breech	11	6 12	Brief period of resuscitation at birth	1	40
4	Diabetes	Normal	3½	5 10	Normal	2	25
5	None	Face presentation	9½	8 2	Pseudocyanosis, due to face presentation	1	70+
6	Banti's disease	Normal	12	9 12	Normal	2	70+
7	None	Normal	17	9 3	Colon-bacillus septicemia; discharged improved on 44th day	25 35	70+ 70+
8	None	Normal	2½	4 4	Three tarry stools on 2nd day; red-cell count 6,000,000, bleeding time 1 minute, clotting time 5½ minutes	2	70+
9	None	Normal	21	9 4	Bloody vomitus at 2 hours, bloody stools at 24 hours; red-cell count 5,300,000, bleeding time 5 minutes, clotting time 3¼ minutes	2	60
10	None	Normal	33	9 0	Coffee-grounds vomitus and tarry stool on 3rd day; red-cell count 5,200,000, bleeding time 2½ minutes, clotting time 2 minutes	3	50
11	Hydramnios	Normal	10	8 15	Catheter resuscitation at birth, old blood in stools at 3 days; red-cell count 3,700,000, hemoglobin 87%, bleeding time 3 minutes, clotting time 4 minutes	4	35
12	Toxemia	Induced	10	6 1	Catheter resuscitation at birth, grossly bloody stools and marked dehydration at 2 days, old intracranial hemorrhage confirmed at 14 days; red-cell count 6,500,000 to 7,200,000, bleeding time 4 minutes, clotting time 5 minutes	3 7 25	25 10 70+
13	None	Normal	24	5 0	Cyanotic attacks for 3 days after birth; bleeding for several hours from heel puncture; red-cell count 6,760,000, bleeding time 2 hours, clotting time not determined	5 24	25 70+
14	None	Normal	?	9 0	Bloody vomitus and stools at 2 days; red-cell count 3,200,000, bleeding time 22 minutes, clotting time 6 minutes; transfusion given	2	20
15	Funnel pelvis	Forceps	21	7 13	Given oxygen at birth; forceps hematoma of jaw, pink-stained vomitus and high-pitched cry at 36 hours; red-cell count 3,500,000, bleeding time 15 minutes, clotting time 3 minutes; on 4th day bleeding time 4 minutes, clotting time 4½ minutes; lumbar puncture showed 540 red cells, mostly crenated	2 4	15 60
16	Prenatal vaginal bleeding	Induced	3½	8 13	Neonatal asphyxia, multiple ecchymoses, bleeding for 7 hours from heel puncture; red-cell count 5,000,000, bleeding time 6½ minutes, clotting time 12 minutes; lumbar puncture suggested intracranial hemorrhage	2 7	5 50
17	Pyelitis	Normal	6	10 6	Red-tinged vomitus at 24 hours, recurring later on second day, heel puncture oozed for 24 hours, red-cell count 5,280,000, bleeding time 9½ minutes, clotting time 13 minutes; transfusion given	2	5
18	None	Normal	?	9 11	Vomiting until 8th day, when operation relieved duodenal stenosis; heel puncture bled for 7 hours on 8th day; bleeding time not determined, clotting time 15 minutes, hemoglobin 80%	8	5
19	None	Normal	3	? ?	Jaundice on 3rd day, spontaneous ecchymoses from 10th day; cord stump bled intermittently after 12th day until transfusion was given on 19th day; operation at 8 weeks disclosed inspissated bile in common duct; red-cell count 1,640,000, bleeding time 30 minutes, clotting time not determined	19	5
20	None	Normal	14	7 7	Vomited all feedings from birth until regulated with thickened feedings at about 1 week; red-cell count 5,400,000 on 3rd day	3 9	15 15
21	Toxemia (mild)	Forceps	5	8 9	Mild cyanosis for 6 hours; marked caput succedaneum	1	25
22	None	Normal	7	9 11	Severe neonatal asphyxia; died in 16 hours	½	30
23	None	Breech	2	7 9	Right monoparesis at 1 day; lumbar puncture (2 days) showed 6000 red cells, all crenated	1 5	20 70+
24	Hypertension in 45-year-old mother	Normal	24	7 14	Neonatal asphyxia with recurrent dyspnea on 2nd day; lumbar puncture showed 18,000 crenated red cells; diagnosis of residual high cervical-cord injury made at 3 months	2 6	15 20
25	Toxemia	Normal	2	8 13	Cyanosis and tense fontanelle on 3rd day; cyanosis still visible on 13th day; lumbar puncture unsatisfactory	6 11	25 50
26	None	Normal	14	9 12	Convulsive episodes from 2nd to 10th day; calcium therapy instituted on 10th day; lumbar puncture unsatisfactory, bloody tap (?)	6	70+
27	None	Breech	38	7 0	Head and shoulder injuries at birth; subdural hemorrhage; left subdural tap at 14 days showed 7200 crenated red cells	34	70+

portion of infants with hemorrhagic disease of the newborn have a definite history of neonatal asphyxia. Clifford[24] has also drawn attention to the significant degree of liver damage found in infants dying with neonatal asphyxia — an observation of particular value in view of the known dependence of the blood prothrombin level on the integrity of the liver.[25] Hence it appears that neonatal asphyxia may in a certain number of cases be closely associated with the appearance of neonatal hypoprothrombinemia, if it is not in fact influential in the production of this condition.

No definite relation between prothrombin titer and clinical hemorrhage was observed. Whereas 8 patients with levels below 35 per cent (range 5 to 25 per cent, average 12 per cent) manifested a hemorrhagic tendency, 8 other patients with subcritical levels (range 15 to 30 per cent, average 22 per cent) showed no tendency to bleed. On the other hand, 4 of the 11 patients with initial prothrombin levels above 30 per cent exhibited melena. This melena, however, was not severe in any case, nor was it accompanied by other signs of a bleeding tendency. By contrast, 7 of the 8 bleeders with prothrombin levels of 30 per cent or less manifested either severe bleeding, a marked anemia or both; and all 7 had either a prolonged bleeding time or else bled for several hours from a skin puncture. In general, it was noted that infants with prothrombin titers below 15 per cent tended to bleed severely; those with titers from 15 to 30 per cent did not always bleed, but when they did, the hemorrhage was usually severe; whereas infants with titers above 30 per cent, though they might bleed, did not manifest more than transitory hemorrhage. Thus hemorrhagic disease of the newborn appears to be a syndrome in which two types of hemorrhagic tendency may be distinguished. There is a mild group having no intrinsic coagulation defect, in which transitory bleeding is probably induced by some factor such as gastrointestinal irritation from the primary bacterial invasion of the digestive tract,[26] vascular congestion or rupture of minor blood vessels during delivery. Severe bleeding, on the other hand, appears to have a close correlation with a low level of plasma prothrombin, and in the series of cases presented showed a close relation to prolonged bleeding time. The latter group might be regarded as having genuine hemorrhagic disease of the newborn, and must be differentiated from the first group in any evaluation of the etiology or therapy of this syndrome.

The very low prothrombin levels observed in Cases 18 and 20, in both of which severe nutritional disturbances were present, may be related to the finding that the maintenance of a normal plasma-prothrombin level, even in an adult, is dependent on adequate assimilation of vitamin K.[27] In this connection, the recent reports of an elevation in the neonatal prothrombin level, following oral administration of vitamin K to newborn infants[28, 29] or to mothers before delivery,[29, 30] are of great physiologic as well as clinical significance.

No correlation could be observed between prothrombin titer and duration of labor. Nor was any relation apparent between prothrombin level and birth weight. We were unable to confirm the correlation reported by Hellman and Shettles[30] between prematurity and low plasma prothrombin.

Summary

Plasma-prothrombin titrations were performed on thirty-nine venous bloods from 27 infants between the ages of three hours and twenty-five days. All but 6 of the infants were studied while under observation for some neonatal disturbance.

Titers ranged between 5 and 70+ per cent of the adult normal. No correlation could be observed between plasma-prothrombin level and birth weight or duration of labor.

Nine of 11 infants observed following neonatal asphyxia or intracranial hemorrhage had prothrombin titers below 35 per cent.

Although no absolute relation was observed between the prothrombin level and the tendency to hemorrhage, it was noted that infants with titers below 15 per cent tended to bleed severely; that infants with intermediate levels (15 to 30 per cent) might or might not bleed — when they did bleed, the hemorrhage was usually severe; and that infants with titers above 30 per cent sometimes manifested a transitory melena, but none in this series exhibited a severe bleeding tendency.

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MEDICAL PROGRESS

NEUROSURGERY

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THERE is evidence from the medical literature of 1940 that neurosurgeons are rapidly becoming more and more aware of the fact that neurosurgery is no longer confined to the field of brain tumors. Indeed, it seems probable that this relatively small and unimportant branch of the specialty will soon be relegated to its proper place and given only such attention as its actual importance justifies. This is not, as some might think, because of the preponderance of "war papers." Actually, these formed only a small part of similar papers on craniocerebral, spinal and peripheral nerve trauma in 1940.

CRANIOCEREBRAL INJURIES

De Martel,[1] before his tragic death, advocated prompt removal of patients with craniocerebral wounds to adequately equipped hospitals in the rear, trephination to expose the internal table of the skull and general use of the bone-flap method of approach to the projectile. Vincent[2] also urges early therapy, something in the nature of débridement with removal of the projectile if possible or, if that is impossible, its removal by way of a secondary approach. He avoids drainage. A review of this subject written from the French and Spanish point of view by Ducuing, d'Harcourt, Griño and Folch[3] covers the same ground in more detail, as does a similar Swiss article by Decker and Rossier.[4]

Outside the field of war wounds, a study of subdural hematomas in patients with mental disease by Allen, Moore and Daly[5] and papers by Joslin[6] and Bréhant[7] on traumatic diabetes are significant.

Allen and his associates found 245 cases of subdural hematoma among 3100 consecutive autopsies on psychotic patients. This is an incidence of 7.9 per cent — a figure that may well cause any physician to reflect before recommending commitment of a patient who has become mentally abnormal following a head injury, but in whom the intracranial pathology has been inadequately demonstrated.

Joslin, disturbed over a paper on diabetes mellitus makes the following statement, — it is quoted because it is an authoritative and, to me, final opinion on this subject at this time, — "So far as I can remember, no definite case in which I considered trauma a cause of diabetes has occurred among the approximately 19,000 patients with diabetes mellitus and glycosuria who have consulted me." From the other point of view, but with much less experience, I can cite the fact that in over 3000 craniocerebral injuries I know of no case in which the trauma had produced either diabetes mellitus or glycosuria by the time

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the patients had left the hospital after treatment for their injuries.

Diabetes insipidus may fall into a different category. The evidence is still too meager to justify a more definite statement, however. So far as it goes in support of trauma as a possible cause of this other type of diabetes, Bréhant, on the basis of one case, states that it cannot be regarded as rare or unusual and then "rings all the changes" on the old theoretical concepts of its cause.

Spine and Spinal-Cord Injuries

Although only remotely connected with the problem of spine and spinal-cord injuries, the papers by Zeligs[8] on congenital absence of the sacrum, by Kuhns et al.[9] — among other subjects — on motion in the vertebral column and by Batson[10] on the function of the vertebral veins and their role in the spread of metastases merit detailed study. I can add another case to Zelig's collection and a note to the effect that, at the same time, there was another child in the wards who had a double sacrum; both cases were seen in consultation with Dr. Louis G. Howard. The importance of Batson's work can hardly be overestimated; any surgeon who deals with malignant tumors and their metastases should make himself familiar with it.

Papers on all aspects of rupture of the intervertebral disk, the protrusion of the nucleus pulposus and its compression of the cord, the cauda equina or the individual spinal roots continue to flood the literature. The best opinion today seems to be that a certain diagnosis of a protruded nucleus can be made only when there is a reasonably connected history of back injury, an increase of total protein content above normal in the specimen of cerebrospinal fluid that was collected closest to the assumed level of protrusion, — other samples having a normal content, — a defect, visualized by x-ray study in the intra-arachnoid column of lipiodol or air with or without a partial block, and finally a demonstration of peripheral neurologic changes that are characteristic of radiculitis and that can be explained only on the basis of an irritation or compression of one or, at most, two adjoining roots. Of all these requirements, the last is by far the most important. Indeed, my experience leads me to believe that when the tumult and the shouting have died down, it will come to be recognized as the one indispensable prerequisite for this diagnosis.

Air or, better still, oxygen is gradually replacing lipiodol as a means of visualizing the outlines of the spinal canal. Chamberlain and Young's[11] paper is most convincing. Poppen[12] reports that "oxygen spinograms on 175 patients suspected of having herniated intervertebral disks were thought to be positive, and later verified by operation, in 150." On the other hand, Hampton[13] reports an accuracy of 93 per cent in 133 cases in which iodized oil was used intrathecally. He strongly favors the use of this medium in preference to oxygen, even though he recognizes that the oil is a foreign body and may act as such in producing disturbing or dangerous effects. In this connection, Garland and Morrissey[14] report finding intracranial collections of iodized oil some years following lumbar myelography in approximately two thirds of a series of 25 cases. However, they found no case in which any symptoms or positive neurologic findings could be ascribed to the presence of the oil. A more widespread use of oxygen or air is undoubtedly retarded by the technical imperfections of the method. These result in underfilling or overfilling of the sac and consequent poor visualizing of the edges of the column of oxygen.

It is still generally agreed that adequate orthopedic treatment should precede the use of laminectomy as a method of treatment of protruded nuclei. If laminectomy is used, the removed bone should never extend beyond one or two adjoining laminae on one side only. Mixter and Barr[15] state that it is necessary to cut away only the upper and lower edges of two adjoining laminae. Hamby[16] describes an interlaminal removal of the protrusion that he believes to be possible in some cases. It appears that, with such minimal bone removal and ligamentous damage, the operative removal of a protruded nucleus does not per se require subsequent fusion of the spine.

An end-result study of the bone lesions accompanying spinal-cord injuries in 76 cases is reported by Munro and Wegner.[17] They point out that the therapy and prognosis of cervical-spine injuries that do not involve the spinal cord or are already chronic when first seen are quite different from those in which the cord injury plays a part and in which the patient comes for treatment immediately following the injury. Treatment in the combined cases is conditioned by the therapeutic needs of the damaged cord and the presence or absence of a spinal subarachnoid block. Fifty per cent of their patients died within the first forty-eight hours, and 45 per cent before discharge and as the direct or remote result of the injury. Abandonment of immediate laminectomy reduced their mortality 30 per cent. Twenty-eight of the 30 living patients had satisfactory end results, and 4 others died of intercurrent disease. These authors believe that the use of plaster-of-Paris casts

in cervical-cord injuries is dangerous and ordinarily contraindicated. In this connection, they[18] have modified and used with satisfaction an adjustable cervical spinal brace. In connection with cervical spinal injuries and perhaps of importance from the industrial aspect is the report by Hanflig and Schlosberg[19] of a rare case of nontraumatic dislocation of the atlantoaxial joint.

General care of spinal-cord injuries. Adequate tidal drainage of the urinary bladder is or should be an essential part of the general care of all spinal-cord and cauda-equina injuries. The original apparatus devised for this purpose was never intended to irrigate the bladder except as a secondary function. Its prime purpose was to permit the surgeon to adjust the intravesical pressure to the type of bladder that the apparatus was serving, and by regular emptying, in addition to a minimum of irrigation with some antiseptic solution, to counteract the deleterious effect of the residual urine that was necessarily always present. The inability to deliver significant quantities of irrigating solution to the bladder has now been overcome by one of the modifications of the apparatus devised by MacNeill and Bowler.[20] This improved tidal-drainage apparatus, as diagramed in Figure 6 of their article, should replace the original in the treatment of so-called "neurogenic bladders." When it is used, however, a change should be made in the irrigating fluid. If a 1:30,000 solution of potassium permanganate is introduced into a bladder in the large quantities that are permitted by this apparatus, either a mucous or alkaline cystitis is set up. In the latter event, calculi will form. These complications can be avoided by the use of 0.5 per cent acetic acid instead of the permanganate solution.

In line with the cystometrograms that are a necessary part of tidal-drainage therapy, White, Verlot and Ehrentheil[21] have adopted the U type of cystometer[22] for use in a study of activity of the colon. They have found that the colon is the physiologic analogue of the bladder. It has the same stretch reflex and the same ability to distend, to act as a reservoir and to initiate emptying contractions in response to a sensory stimulus caused by a given amount of fill. Its activity is governed by a spinal reflex, and emptying contractions may be inhibited but not initiated by impulses from the cerebral cortex.

With the overlapping of neurologic into genito-urinary surgery, an authoritative statement concerning the use and abuse of the urethral catheter will be welcomed by neurosurgeons. This is found in Quinby's article.[23] The bacteriology of urinary-tract infection is well covered by Scholl et al.,[24] and the present status of urinary antisepsis by Culver and Seifert.[25] An interesting case of neurogenic dysfunction of the bladder due to spinal anesthesia is reported by Peirson and Twomey.[26]

Peripheral-Nerve Injuries

With the advent of war, articles about peripheral-nerve injuries again begin to appear in the literature. That war is not necessary to the proper understanding of perhaps the most important of all peripheral-nerve problems is evidenced by the article on the traumatic lesions of the nerves of the wrist and hand by Dr. Torr Wagner Harmer.[27] This classic was published shortly after his death and may well serve as a fitting memorial to him. In it the reader will find the last word on this subject.

From Germany comes an article by Ehalt.[28] He cites an experience with 77 cases of nerve injuries and advises that in cases of open nerve injuries immediate suture is required, but that in closed nerve injuries operation should not be performed until six months have elapsed, since the outlook for recovery is very favorable. Leriche[29] calls attention to the variability of results and their dependence on the particular nerve involved. He believes that traction on peripheral nerves prevents good healing. The best work comes from England. Three articles in the *Lancet* should be read in detail by all who are interested in this problem. One is by Cairns and Young[30] on the whole subject of treatment of gunshot wounds of the peripheral nerves; another is by Young and Medawar[31] and concerns fibrin suture of peripheral nerves, with particular reference to the measurement of the rate of regeneration; and the third is by Young, Holmes and Sanders[32] on the importance of the peripheral stump and the value of nerve grafts in nerve regeneration.

Finally, a paper by Homans[33] should be mentioned because of its importance as explaining one of the causes of hitherto unexplainable pain in the extremities. He speaks of it as "minor causalgia" and defines it as a "hyperesthetic neurovascular syndrome." It comes about in patients in whom probably "there is something basically at fault about the nervous system." Abnormal and painful neurovascular reflexes involving the sympathetic fibers in the vessels of the extremity and their connections with the central nervous system on the one hand and the small vessels on the other are initiated by apparently trivial peripheral stimuli. Fortunately these painful symptom complexes can usually be relieved by sympathetic-nerve block. Characteristic changes in the texture, general appearance, color and often temperature of the skin in the painful area occur. The severer forms are associated with local injury and may be very chronic.

Epilepsy

Gibbs[34] published in December, 1939, an extremely important paper on electroencephalography in epilepsy. In this study he presented clearly the present concept of epilepsy as exemplified by electroencephalographic and other associated diagnostic studies. From this work it is apparent that "fits" do not accompany all epileptic attacks, that there is a definite hereditary factor in the disease and that pathways of new investigation have been so opened out that one can say with the author that electroencephalography "has revolutionized the study of epilepsy." The following April, Lennox,[35] in a more general paper on the treatment of epilepsy and migraine, re-expressed the essentially diagnostic data published by Gibbs and added certain other facts concerning the relation between this disease and migraine and the various available methods of treatment with their advantages and drawbacks. No pediatrician, general practitioner, neurologist or neurosurgeon should fail to read these two papers in detail; no one today can either properly diagnose or properly treat epilepsy unless he is thoroughly familiar with their contents.

Miscellaneous

Bedsores are a source of trouble and annoyance to nurses, patients and doctors alike. This is especially true in association with spinal-cord injuries. Munro[36] has presented evidence to show that in spinal-cord injuries there is reason to believe that all bedsores are preceded by pressure sores, which in turn develop because of too prolonged pressure on bony prominences, maceration of the skin, with destruction and removal of the protective horny layer, and local infection. In the presence of spinal-cord injury that destroys motor reflex activity — that is, spinal shock — and particularly in association with thoracic-cord injuries, the peripheral vasomotor reflexes cease to act. This causes spreading radial necrosis and the characteristic bedsore, as contrasted with the pressure sore. The treatment recommended is to move such patients once an hour; to prevent the development of any serious exhaustion by appropriate feeding, transfusions, administration of vitamins and the like; to prevent the occurrence of general sepsis and to maintain a constantly dry bed by the proper use of a tidal-drainage apparatus; and to avoid all forms of external artificial splinting or support to the spinal column so long as the patient with a spinal-cord injury is bedridden. This therapy is especially important in the thoracic-cord injuries. In connection with this work, it was noted that the patients had a severe hypoproteinemia, and that pressure sores and bedsores that were already established could be healed only by the feeding of very high protein diets. Ravdin,[37] Elman,[38] Holman[39] and Koster and Shapiro[40] all write on various aspects of this problem, although not directly about bedsores or pressure sores. Brooks and Duncan[41, 42] contribute significant data on the effects of temperature on the survival of anemic tissue, and the effects of pressure on tissues in general.

Shuman and Jeghers[43] report their findings in relation to the value of routine blood-protein determinations in 320 consecutive cases.

Crandon, Lund and Dill[44] report on experimental human scurvy, and Lund[45] on the effect of surgical operations on the level of cevitamic acid in the blood plasma. Along this general line Elkinton, Gilmour and Wolff[46] contribute a useful paper on the control of water and electrolyte balance in surgical patients, and Maddock and Coller discuss the sodium chloride metabolism[47] and the water and electrolyte balance[48] in connection with surgical problems. Finally, Drew, Scudder and Papps[49] describe four simple tests by which data can be obtained in an emergency to determine the degrees of water and of water plus protein loss, to anticipate the onset of shock and differentiate it from simple circulatory collapse, to detect dehydration and to direct treatment more rationally for the alleviation of any of these conditions.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27181

PRESENTATION OF CASE

A fifty-eight-year-old Russian-American entered the hospital complaining of weakness and dizziness.

The patient had been a hospital employee for twenty-five years, and four years before admission a routine physical examination was negative except for a rudimentary left ear, left palatal and facial weakness, with inability to close the left eye, and slight right torticollis; these defects had been present for as long as the patient could remember. Three weeks before admission his friends noticed that he was forgetting things, and he complained of slight weakness and dizziness. Ten days before entry the patient came to the Emergency Ward complaining of headache, wobbliness and weakness. He appeared "washed out," and his tongue was coated, but the examination was otherwise negative, the temperature being 97°F. and the pulse 88. The patient was ordered off work for four days, but at the end of that time he was suffering from headache, dizziness, fatigue, weakness, anorexia, generalized muscle pains, nausea and vomiting. A diagnosis of grippe was made, and he entered the hospital for two days; he then remained at home in bed for two days, after which he returned stating that since discharge he had been so weak that he fell when attempting to walk and was dizzy even when lying down.

The past illnesses and family history were irrelevant.

On examination the patient was well developed and well nourished, but vague and unable to cooperate. The left external ear and auditory meatus were absent. The pupils were small, but equal, the retinal vessels tortuous, and he was unable to count fingers accurately; it was impossible to make out the visual fields. A peripheral seventh-nerve palsy was present on the left, as had been noted four years previously. All four extremities were spastic, slightly more so on the left; there was no paralysis, but a definite impairment in the performance of fine movements. The tendon reflexes were equal but exaggerated, with ankle clonus and a Babinski sign bilaterally.

The tongue was dry and coated. Examination of the heart, lungs and abdomen was negative; the blood pressure was 120 systolic, 70 diastolic; the peripheral vessels were hard and tortuous, and the legs cold and mottled.

The temperature, pulse and respirations were normal.

The urine was normal. The blood showed a red-cell count of 5,300,000 with a hemoglobin of 16.5 gm. (photoelectric-cell technic), and a white-cell count of 8500. The nonprotein nitrogen of the blood serum was 18 mg. per 100 cc., and the blood Hinton reaction was negative. The stools were normal.

On lumbar puncture the initial pressure was zero; the fluid was slightly yellow; the white-cell count was 1, and the red-cell count 520 (cells not crenated), per cubic millimeter; the protein was 174 mg. per 100 cc.; the gold-sol curve was 2544521000; and the Wassermann reaction was negative.

On the fourth hospital day the patient was less confused. There was weakness of all movements of the left arm and leg, with a more marked spasticity in these limbs. The tendon reflexes were more active on the left side. Tactile fremitus was increased over the right lung base, with many coarse rales over the area. An x-ray film of the chest, taken three days later with a portable machine, showed ill-defined consolidation in both lower-lung fields, as well as in the medial portion of the right upper-lung field. The patient gradually weakened, lapsed into coma without developing further neurologic signs, and died two weeks after admission, the temperature rising to 103°F. three days before death.

DIFFERENTIAL DIAGNOSIS

DR. JOHN H. TALBOTT: This patient was fifty-eight years of age and had presumably always carried stigmas acquired in utero or at an early age. The rudimentary left ear, the left palatal and facial weakness and the right torticollis are of historical interest but are not pertinent to the discussion of the present complaint. All the presenting symptoms, as well as those which appeared during the three-week period of observation, indicate an acute and rapidly progressive process. His first complaint, which was called to his attention by his friends, was a poor memory. He had, in addition, headache, weakness, anorexia, nausea and vomiting, and dizziness. These symptoms, together with a subnormal temperature of 97°F., suggest an acute attack of adrenal insufficiency in an advanced stage of Addison's disease. The description of the weakness is particularly suggestive

of Addison's disease. Patients with this malady may fall to the floor in a heap after one or two attempted steps. There is little else to suggest a diagnosis of an Addisonian crisis, however, and I shall not consider it further.

On examination the patient was well developed and well nourished. This is presumptive evidence against certain types of cancer, either primary or metastatic. He did not have, I should say, carcinoma of the stomach with metastases to the central nervous system. Anorexia, nausea and vomiting might accompany carcinoma of the stomach, but if this disease had been responsible for the symptoms I doubt if the patient would have been described as well developed and well nourished. The first positive finding, which undoubtedly is pertinent to the discussion, is the presence of generalized spasticity. Although all the lower extremities were spastic, the left arm and leg were more so. A Babinski sign was observed bilaterally, as well as ankle clonus. These findings point to some disturbance of the motor tracts involving the right as well as the left side of the body. I should doubt if the lesions were as high up as the motor cortex, and I am inclined to place them at the site of decussation of the pyramidal tracts or lower. There is no mention made of any sensory changes, although it might have been difficult to collect precise data in a patient who had obvious central-nervous-system disease. I thought, in passing, of pernicious anemia to explain the generalized spasticity, but the red-cell count of 5,000,000, as well as a normal white-cell count, pointed against a diagnosis of Addisonian anemia. The white-cell count is also against an acute infection, such as multiple cerebral abscesses, to explain the picture. The nonprotein nitrogen was normal, and we cannot explain the nervous changes on the basis of renal insufficiency and uremia. No mention was made of any intoxication or self-administered poison, and we are forced to dismiss this possibility without further discussion. The blood Hinton reaction was negative, as was the spinal-fluid Wassermann reaction. One thinks of cerebrovascular syphilis in such a clinical picture as this, but again we must dismiss the possibility for want of more evidence. The record tells us that the initial pressure during a lumbar puncture was zero. Was that repeated? Was any statement made in the record about this interesting point?

Dr. Charles S. Kubik: I do not believe that it was.

Dr. Talbott: It is unusual to get a lumbar-puncture pressure of zero without commenting on it.

Dr. Kubik: It is.

Dr. Talbott: I wonder whether this was a valid observation, but since it was not repeated, we must accept it at its face value or even disregard it if we choose. The spinal fluid was slightly yellow, and the cell count was 1 white cell and 520 red cells per cubic millimeter. Since the red cells were not crenated, it is possible that they were introduced at the time the tap was performed and were not present in the spinal fluid before the tap. We cannot, however, attribute a spinal-fluid protein of 174 mg. per 100 cc. to trauma. If one considers the amount of protein that accompanies a red-cell count of 520, there is still a wide gap between this value and 174 mg. The presence of slightly yellow fluid is additional evidence in favor of previous mischief in the central nervous system.

I should like to see the x-ray films at this point.

Dr. George W. Holmes: We have only one film, and that was taken with a portable machine. Apparently the patient was rather sick. The evidence is not complete. I hope it will not confuse rather than help you.

In this film the right lung field looks smaller than the left. I do not think you can rely on this finding, but I think you should consider it.

Here near the root of the right lung is a dense poorly defined shadow, which could be interpreted as a mass. The heart shadow is within normal limits. There is no evidence of fluid in the pleural spaces. There are also changes in the lower part of the right lung. One way to interpret it would be to say that this mass at the root of the lung accounted for the decrease in the size of the lung and the changes in the lower part of the chest, and that it was causing interference with passage of air into the lung. Another way to interpret it is to say this was part of a bronchopneumonic process, and that the mass was caused by enlarged lymph nodes.

Dr. Talbott: Are you able to exclude a large pulmonary infarct or multiple infarcts?

Dr. Holmes: I think that is unlikely. It could be an acute respiratory infection, or it could be a tumor.

Dr. Talbott: If it is tumor, does it look more like primary bronchiogenic carcinoma, a mediastinal lymphoma or a Pancoast tumor?

Dr. Holmes: If it is tumor, it is a primary bronchiogenic tumor.

Dr. Talbott: It is not infrequent to see patients admitted to the hospital complaining of cerebral symptoms who have perfectly negative histories of disturbance in the lungs, yet we may find a primary bronchiogenic tumor that has metastasized to the brain. I recall very vivid-

ly one patient known to most of us a decade ago who had essentially that story. His presenting complaints on admission to the hospital were cerebral and not pulmonary. There was nothing in the history to suggest a primary tumor in the lung. Following a cerebral exploration a metastatic lesion was discovered, and it remained for the pathologist to tell us that it was probably primary in the bronchus. Another point in favor of a primary bronchiogenic carcinoma in the patient under discussion is the statement regarding nourishment and development. I believe that if this patient did have a tumor with metastases it was consistent only with a bronchiogenic carcinoma.

Before I came to this conference, I was inclined to put primary bronchiogenic carcinoma as a second possibility, and believed that the patient's symptoms were probably due to a cerebral vascular accident. Multiple thromboses of the cerebral vessels could give most of the findings described. With this syndrome there may be a story of forgetfulness, weakness and dizziness, and the course may be as rapid or more rapid than that observed here. It could explain everything up to the rise in temperature three days before death. If we assume the primary cause of death was cerebral thrombosis, then we can attribute the terminal rise in temperature to an infection in the lungs, or better to a pulmonary infarct, which is usually not recognized as a terminal event. In conclusion, I propose to adhere to my preconference diagnosis of cerebral thrombosis and put down as a second possibility, primary bronchiogenic carcinoma with cerebral metastases.

Dr. Edward F. Bland: May I ask for information about the lumbar puncture? Does that mean block above the site of puncture?

Dr. Kubik: We occasionally find a low spinal-fluid pressure in cases with increased intracranial pressure. We see it rather rarely, and usually after a previous lumbar puncture, but here it was observed at the first puncture. In some of these cases we find a herniation of the brain through the tentorium, with flattening and obstruction or partial obstruction of the aqueduct. With such a condition any escape of spinal fluid before the manometer is connected to the needle may cause the pressure to drop rapidly.

Dr. Bland: What does jugular compression do in such a situation?

Dr. Kubik: One should not try jugular compression in a situation like that. There would probably be a little rise in the spinal pressure. It might also result in death from respiratory failure.

What do you really think about the films, Dr. Holmes?

Dr. Holmes: I do not know. It would just be a guess. There is insufficient evidence, and the examination was incomplete.

Dr. Kubik: What would you guess?

Dr. Holmes: If I had to make a guess, I should agree with Dr. Talbott.

Dr. Augustus S. Rose: If it is a brain metastasis, would Dr. Talbott make a statement as to where he thinks it was?

Dr. Talbott: It was bilateral. If cerebral thrombosis is the correct diagnosis, then there might have been several areas of softening in the brain stem. Certainly the lesions must have been below the motor cortex.

Dr. Rose: There is no definite statement about papilledema.

Dr. Talbott: I did assume, however, that there was no increased intracranial pressure. No mention was made of retinal exudates, and there was no good description of the retinal vessels. One sentence included in the abstract is difficult to interpret: it is mentioned that it was impossible to make out the visual fields. Does this mean that the patient could not co-operate, or that the visual fields were so restricted that they could not be determined accurately?

Dr. Kubik: I should think the former.

Dr. Talbott: That is what I assumed.

Dr. Kubik: Sometimes the examiners are too easily discouraged.

Clinical Diagnosis

Cerebral accident.

Dr. Talbott's Diagnosis

Cerebral thrombosis, with pulmonary infarcts,
Or primary bronchiogenic carcinoma, with metastases to the brain.

Anatomical Diagnoses

Bronchiogenic carcinoma of the lung, with metastases to the brain.
Agenesis of the left middle ear and congenital deformity of the left auricle.
Pleuritis, fibrous.
Anthracosis, marked.

Pathological Discussion

Dr. Kubik: This was a bronchiogenic carcinoma of the left upper-lobe bronchus, measuring about 7 cm. long and 4 cm. in diameter. Metastases were present only in the brain, and those, as usually happens, were multiple. The largest

one, measuring between 4 and 5 cm. in diameter, was in the right occipital lobe. There were smaller ones near the upper part of the motor cortex on the right, in the right temporal lobe and in the cerebellum, very near the dentate nucleus, which may, I suppose, have accounted for some of the dizziness. There was some herniation of the hippocampal gyrus through the tentorium. This probably accounted for the low spinal-fluid pressure.

The shadow seen in the x-ray film, on the right side, represents enlarged lymph nodes, in which, however, there were no metastases. The tumor itself is not visible in the film.

DR. HOLMES: I have been told by a member of your department that metastases below the tentorium are very uncommon. Do you agree with that?

DR. KUBIK: No; they are found above and below, often in the same case.

DR. HOLMES: So many of these people with bronchiogenic carcinoma die of respiratory failure that I was wondering if the metastasis to the base of the brain was the original cause of that.

DR. KUBIK: I can recall several cases with cerebellar metastases, or cases in which there were both cerebral and cerebellar lesions, those in the cerebellum being much larger and chiefly responsible for the patient's symptoms. I should agree so far as to say that metastases to the forebrain are commoner.

CASE 27182

PRESENTATION OF CASE

A forty-five-year-old French-Canadian housewife entered the hospital complaining of nausea and epigastric pain.

Twenty-five years before admission, the patient began to suffer from attacks of nausea and vomiting that lasted several days and recurred every few months. These attacks were relieved by the induced vomiting of a sour, yellow fluid. Between times her appetite was good, but she disliked fried food and sweets and believed that they initiated the attacks. Nine years before entry, recurrent attacks of moderately severe, sharp, nonradiating, high epigastric pain appeared, occurring after meals and lasting but a few minutes. Four years before admission, the patient suffered a very severe knifelike, mid-epigastric pain, which radiated to the back and was associated with nausea and vomiting. This attack lasted four days and then subsided, but recurred two years later, when it lasted one day. At another hospital, three months before admission, a laparotomy was performed for an intended cholecystectomy, but a small fairly well-circumscribed mass was found on the anterior wall of the stomach near the pylorus, and another mass was noted on the dome of the right lobe of the liver. The gall bladder was said to have been filled with stones, but a cholecystectomy was not done. Because the patient had not been prepared for gastric resection, only a biopsy of the gastric tumor was performed; the specimen was reported as a slowly growing adenocarcinoma. One month before admission the patient noticed that for two days her stools were yellow; at that time she entered still another hospital, where she remained until admission here. X-ray examinations at the above institution were essentially normal, and no gastric abnormality could be demonstrated, except for pylorospasm.

At no time during this illness had jaundice been noticed, although it had been present on one occasion when the patient was ten years old; no change in the color of the urine had ever been noticed. The patient had always been mildly constipated and frequently used laxatives. She had lost 20 pounds in the year prior to operation, but had gained 7 pounds since operation.

The patient's father had died of cancer, and "numerous" aunts and uncles on her father's side had succumbed to this disease.

The patient was well developed and moderately obese. Examination was essentially negative except for a healed right-rectus epigastric surgical incision; the blood pressure was 130 systolic, 70 diastolic.

The temperature, pulse and respirations were normal.

The urine was normal. The blood showed a red-cell count of 3,400,000 with a hemoglobin of 75 per cent, and a white-cell count of 6800. The nonprotein nitrogen of the blood serum was 17 mg., the protein 6.6 gm. per 100 cc. and the chlorides 101.8 milliequiv. per liter. A blood Hinton reaction was negative; the serum van den Bergh was normal. A gastric analysis showed no free hydrochloric acid, even after histamine.

A gastrointestinal series on two occasions failed to show evidence of tumor or ulceration in the stomach. The prepyloric area was slightly elevated, possibly owing to postoperative adhesions.

A Graham test showed a faint shadow of the filled gall bladder, superimposed by the hepatic flexure. There were areas of decreased density in the region of the gall bladder, which were either stones or superimposed intestinal gas, more likely the former. The patient was unable to retain the fatty meal.

At gastroscopy a normal peristaltic wave was seen repeatedly, passing over the antrum to a tight closure, presumably the pylorus. No abnormality was visible in the antrum or pylorus. Throughout the body of the stomach there was slightly increased reddening, especially on the crests of the rugae. There was no visible verrucous appearance, ulceration or neoplasm.

A laparotomy was performed one week after admission.

Differential Diagnosis

Dr. Myles P. Baker: Perhaps we might look at the x-ray films first.

Dr. James R. Lingley: Dr. Richard Schatzki performed the two gastrointestinal examinations, and since he had been warned that carcinoma of the stomach had been found at a previous operation you may be sure that he made the examination with great care. He was, however, unable to detect anything wrong with the stomach. As stated in the record, the antrum appears to be slightly displaced upward, and there is a suggestion of pressure defect here, all of which can be attributed to the previous operation. The Graham test showed a poorly functioning gall bladder, a very pale shadow with areas of rarefaction, which were quite sharply defined, round and quite consistent with gallstones.

Dr. Baker: Have you any comment, Dr. Benedict?

Dr. Edward B. Benedict: I had a good look at this patient's stomach. She co-operated well. I could see the antrum and the body all the way around the stomach and could watch the peristaltic waves to the pylorus, but I could see no intragastric lesion.

Dr. Baker: I am assuming that two facts are true. One is that the biopsy diagnosis of adenocarcinoma was correct. I am also assuming that, if the surgeon were able to take a biopsy specimen from a mass described as being on the anterior wall of the stomach near the pylorus, the mass would be in the nature of a fairly large peritoneal implant. Certainly it was quite a superficial mass. We have no evidence here that the carcinoma projected from the gastric mucosa, for there was no evidence by x-ray or gastroscopy of a demonstrable mass. I should have expected that one of these procedures would have revealed a tumor, were its origin in the gland-bearing tissue of the gastric lining. It is unusual for a peritoneal implant to invade the stomach wall and cause ulceration and hemorrhage, but of course it sometimes does.

I think the description of the location of the mass and the subsequent negative findings are more in keeping with the possibility of a secondary adenocarcinoma originating elsewhere. We have no evidence that it was primary in the colon. Presumably a barium enema was given on the second hospital admission. We have no evidence of pelvic neoplasm. The question comes up whether in the presence of known gallstones this patient could have had an adenocarcinoma primary in the gall bladder. It is true that three out of four cases of primary carcinoma of the gall bladder develop in gall bladders that contain stones, as this woman's did. Jaundice and a palpable tumor, which are commonly found with carcinoma of the gall bladder, are not present in more than 50 per cent of the cases. One could have a small carcinoma of the gall bladder and have symptoms only from metastatic disease, or of course from the associated gallstones. On the other hand, metastases from gall-bladder carcinomas are generally characterized by direct extension to the liver, and the regional lymph nodes are involved early. I think such lymph nodes would have been palpable at the previous operation, three months prior to entry here. Peritoneal metastases from gall-bladder carcinomas develop later.

What then of the possibility of adenocarcinoma originating in the body of the pancreas, which is said to develop in cases of chronic pancreatitis and may be associated with gallstones of some duration? Weight loss, which was rather prominent in this case without any story of persistent indigestion, and repeated attacks of pain sufficient to cause lack of appetite and weight loss are prominent features in the progress of cases of carcinoma of the pancreas. Abdominal pain such as this patient must have had to induce decision for surgery for gallstones can occur with carcinoma of the body of the pancreas. If that diagnosis is correct, some of the pain might have been due to the pancreatic lesion rather than to the gallstones. Metastases from such a growth are characteristically spread directly along the peritoneal surfaces from the body of the pancreas. Any tumor in the body of the pancreas could not be very distant from the peritoneal surface, and I think there is a real possibility that this tumor originated in the pancreas with metastasis to the liver and to the anterior wall of the stomach. The description, "a slowly growing adenocarcinoma," is in keeping with such a tumor. The achlorhydria is not inconsistent with such a diagnosis. About one out of six or eight cases of carcinoma of the body of the pancreas without jaundice shows achlorhydria and an anemia of varying character. I can draw no conclusions from the yellow stools mentioned here.

I am more interested in knowing whether the patient had occult blood in the stools. We have no mention of that, so that we are not sure why this patient was anemic.

Dr. Tracy B. Mallory: I am sure occult blood was searched for and not found in the other hospital. In our laboratory one stool was guaiac negative.

Dr. Baker: That does not change my diagnostic impression. I know it is possible that x-ray examination may miss a gastric tumor, but I should not expect it to miss an adenocarcinoma. Pressure defects in the region of the pylorus are not uncommonly found when the tumor is primary in the body of the pancreas.

Dr. Mallory: Dr. Sweet, will you tell us what you found at operation?

Dr. Richard H. Sweet: We were not confronted with the necessity of committing ourselves definitely, as Dr. Baker has been this morning, so that we performed the operation with the idea of finding out what was the matter with the patient. We were impressed with the history that the previous surgeon had found a tumor of the liver, and assumed that it might be a metastatic growth. The first thing I did on entering the abdomen was to expose and take a biopsy specimen from this liver tumor, which was nodular and rather firm. It was located in the dome of the right lobe and had the characteristic appearance of a hemangioma. The Pathology Laboratory confirmed the diagnosis. The patient had gallstones, which were easily felt. The tumor in the region of the antrum was an interesting one. It measured 3 cm. in diameter. It did not feel like carcinoma; its consistence was not uniform, parts of it being firm, and other portions soft. It was attached to the first portion of the duodenum, as well as to the antrum of the stomach, and it felt to me, as I was resecting it, more like a leiomyoma or neurofibroma of the wall of the stomach. It was covered by adhesions, which had resulted from the previous exploration. It was attached to the anterior wall of the stomach, but seemed to come off from the inferior margin of the duodenum and antrum and lay in a hollowed-out depression in the head of the pancreas. Having had the biopsy report from the other hospital of "a slowly growing adenocarcinoma," we assumed that it would be best not to biopsy the tumor again, and I merely did a resection, thinking that by cutting into it I should be spreading about a malignant tumor, although, as I have said, it was a peculiar feeling tumor. I wished after I finished the operation that we had called for the slides from the hospital where the patient had been operated on before.

Having done a radical resection, we had only a few moments left to turn our attention to the cause of the symptoms, namely, the gallstones. The patient had had characteristic gallstone attacks. I therefore opened the gall bladder, scooped out the stones, which were rather large, and closed the gall bladder and then the abdomen. She has since gone home in a state of good health.

Clinical Diagnoses

Cholelithiasis.
Gastric tumor.

Dr. Baker's Diagnoses

Cholelithiasis.
Carcinoma of pancreas, with metastases to liver and peritoneum.

Anatomical Diagnoses

Cholelithiasis.
Ectopic pancreas.
Hemangioma of liver.

Pathological Discussion

Dr. Mallory: Dr. Baker and the clinicians in the hospital here made the natural assumption that, when a biopsy had been done and carcinoma reported and when that appeared to be substantiated by the presence of a metastatic nodule in the liver, there could be no doubt that the patient had cancer somewhere. That, however, was not true. As you have already heard from Dr. Sweet, the nodule in the liver was a hemangioma, an extremely common tumor in the liver, present in more than 2 per cent of all autopsies. The nodule in the stomach and duodenal wall showed on cut surface finely lobulated tissue, which was quite obviously pancreatic even on gross examination. A frozen section readily confirmed that. Nodules of ectopic pancreatic tissue are very common in the duodenum and by no means rare in the stomach. If they happen to be in or close to the pylorus, which is the common location, they may cause a little obstruction. More often, however, they are present without causing any symptoms, which I think was the case here.

The original microscopic slides from the other hospital were finally obtained and reviewed, and it was perfectly obvious in retrospect that they showed pancreatic tissue, not carcinoma. These cases are not extremely common, and I think a good many pathologists, particularly those in small hospitals, may never have seen a case. It is a natural mistake to make, although I think in any of the large hospitals the true character of the tissue would have been recognized even from a small biopsy specimen.

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ANNUAL MEETING

The program arranged for the annual meeting on Wednesday and Thursday, May 21 and 22, at The Copley-Plaza, Boston, and reprinted in this issue of the *Journal*, should interest every member of the Massachusetts Medical Society.

Briefly, the two-day program is divided into the following: papers of general interest on the mornings and afternoons of both days — those on Thursday morning constitute a symposium on hormones, and those on Thursday afternoon, one on war medicine; seven sectional luncheon round-table discussions on Wednesday, for which reservations should be made immediately; and the two annual lectures — the Shattuck Lecture, delivered by Dr. Alton Ochsner, of New Orleans, on Wednesday evening, and the Annual Discourse, delivered by Dr. A. Warren Stearns on Thursday following the annual meeting of the Society. A group of papers on vitamin therapy and one on intestinal obstruction should prove to be particularly valuable, and the talks by Dr. Frank H. Lahey, president-elect of the American Medical Association, and by Dr. Philip D. Wilson, of New York City, should be very enlightening. As in previous years, there will be a continuous motion-picture program; many of the films are in color, and a few are accompanied by sound effects.

The ladies' program will open with music and tea at the Gardner Museum on Wednesday afternoon, followed by the annual dinner at the Ritz Roof, where entertainment will be provided by an exceptionally good floor show. On Thursday, there will be a trip to the South Shore. Following visits to the Dorothy Quincy House and the Adams Mansion in Quincy and to the Old Ship Church and the Cushing Homestead in Hingham, luncheon will be served at Hugo's in Scituate. The trip back to Boston will be made along the famous Jerusalem Road.

The Committee of Arrangements and the Ladies' Committee are to be congratulated for devising well-balanced, comprehensive and interesting programs. The attendance at the one hundred and sixtieth anniversary of the Massachusetts Medical Society should be record-breaking.

SALE OF ALCOHOL IN MASSACHUSETTS

There is a law in Massachusetts that permits the unrestricted sale of 190-proof grain alcohol over the counter by druggists "for mechanical, chemical and medicinal purposes only," without a physician's prescription. From the nature of the statute it is evident that a buyer may use this alcohol for a beverage if he so wishes. The inadequacy of this law has sounded severe repercussions in Massachusetts. In Boston the death rate from alcoholism has increased considerably since the repeal of prohibition. In New York, where grain alcohol is available only on a physician's prescription, the death rate from alcoholism has decreased sharply since the repeal of prohibition.

Massachusetts is the only state in the Union where the unrestricted sale of grain alcohol is permitted.

The grain-alcohol problem asserts itself in the following manner. Although a person dilutes the alcohol with cheap wines and other beverages at the beginning of the alcoholic episode, the subsequent dilutions are apt to grow progressively weaker, and the blood-alcohol level may rise to a point incompatible with life.

The serious problem of alcoholism in Massachusetts has long been recognized. For example, Massachusetts ranks third among the states in incidence of mortality from alcoholism, according to Schmid.[1] Alexander, Moore and Leary[2] report that 4505 deaths certified by medical examiners in Massachusetts from 1928 to 1938 were directly related to the ingestion of ethyl alcohol; they also found that more deaths from alcoholism occurred in Massachusetts than from all other toxic substances combined. Dayton[3] has recently revealed that, of 56,579 first admissions to Massachusetts mental hospitals between 1917 and 1939, 32 per cent of male patients were classified as intemperate and that alcoholic psychoses ranked third among the causes of admission to those institutions in 1938. Kolb,[4] with the aid of the police departments and departments of health of several large eastern cities, showed that in 1939 the number of arrests for intoxication and of the deaths due to alcoholism per 100,000 population, respectively, were as follows: Boston, 5171 and 16.4; Baltimore, 331 and 1.9; New York, 112 and 4.5; Philadelphia, 1608 and 2.4; and Washington, 2987 and 3.9.

On March 20, 1941, a bill was heard before the Committee on Legal Affairs of the Massachusetts legislature that would greatly alleviate the problem of alcoholism by requiring a physician's prescription for the sale of grain alcohol. It received its only opposition at the hands of a paid druggists' lobbyist. It is essential that this bill should be reported favorably by the Committee on Legal Affairs. If it is to succeed, it must be wholeheartedly supported by the Massachusetts Medical Society, and each member is urged to write to his senator and representative.

References

1. Schmid, C. F. Mortality from alcoholism in the United States. *Quart. J. Studies on Alcohol* 1:432-441, 1940.
2. Alexander, L., Moore, M. and Leary, T. Deaths from poisoning: incidence in Massachusetts. 13 pp. Preprinted from *Am. J. M. Jurisp.*
3. Dayton, N. cited in: Alcoholism and the alcoholic: a brief report of a three-day symposium conducted by the Research Council on Problems of Alcohol at Philadelphia, Pa., December 27-29, 1940. *Ment. Hyg.* 25:322, 1941.
4. Kolb, L. Alcoholism and public health. *Quart. J. Studies on Alcohol* 1:605-621, 1941.

OBITUARY*

LLOYD VERNON BRIGGS

1863 – 1941

Lloyd Vernon Briggs died at Tucson, Arizona, on February 28, 1941, in his seventy-eighth year. Such is the record of a simple fact. Not so simple is the task of portraying the personality of this man, a personality that impinged on the professional and public life of the State of Massachusetts, and ultimately made him the motivating and directing force behind a social organism that grew to proportions attracting the rest of the country, and, in fact, the rest of the world, for advice and consultation.

Dr. Briggs was born in Boston, but his roots went back into the old Plymouth Colony. Going back to many of the families that settled in and about Scituate and particularly at Hanover, his ancestry is a fair catalogue of the vigorous persons who, by growing strong themselves in that territory, helped to make Massachusetts strong. With all his other interests, very much in the foreground were affairs having to do with Hanover and its history. Among his earliest publications were historical treatises, of which the record of shipbuilding on the North River is preeminent. His interest was not archaeological, genealogical or even historical — it pervaded all these fields. Never an ancestor worshipper, he had felt himself to be a part of that great living organism that started so humbly three hundred years ago and developed into a substantial fraction of the nation.

No one could think of Dr. Briggs without envisioning old houses and old furniture. His eye caught the unique as well as the beautiful, and long before such collecting had become fashionable, he and his wife had garnered many precious things, not only from their own families, but here, there and everywhere. This interest and evidence of good taste reached its superlative in the house at 64 Beacon Street, though the houses at Hancock, New Hampshire, and Hanover, Massachusetts, were close rivals. So much fine furniture and so many fine furnishings seem never to have been associated with one family.

*The following statement was adopted and placed on the records of the New England Society of Psychiatry and Neurology, at its meeting at Newtown, Connecticut, April 10, 1941.

When yet a boy, Dr. Briggs made a trip in a schooner to what were then the Sandwich Islands, and took part in vaccinating the native population. Incidentally, he made friendships that lasted throughout his life. From this time on he seems to have been a doctor, taking courses here and there, and especially at Tufts, Dartmouth and Harvard, but continually occupying himself with medical matters. For a few years he was concerned with the care of patients in various private sanatoriums in Massachusetts. He soon assumed responsibility for patients himself, traveling about with them or caring for them at Hancock or Hanover. In 1899 he took his medical degree from the Medical College of Virginia at Richmond. He then started active practice in psychiatry in Boston. For a while his private patients absorbed his attention, but soon the public care of the insane became the ruling passion of his life.

He soon interested himself in the Massachusetts institutions. These institutions were unquestionably good at that time, and yet there were new things to be done. A liberalizing movement was on foot, pervading all human affairs. Certain old abuses had to be abolished, and certain new technics had to be installed. Massachusetts institutions had grown up and flourished under a system of trusteeships. These called on the best in the community for gratuitous service, and the record of one hundred years or more is undeniably good. But the germ of centralized public control had arrived, and the gradual assumption of responsibility by a state board could no longer be resisted. Beautiful as the old and perhaps more benevolent attitudes were, efficiency and standardized effort were in the air. Dr. Briggs put his heart and soul into this, allying himself with many of the leading figures in America who were doing similar work in other states. One need not dwell on the bitterness aroused. It is enough to exult in the achievement of the construction of the present Massachusetts Department of Mental Health, with a single directing head. Dr. Briggs did this almost alone, and it is only fair to say that almost alone he selected the late George M. Kline as the new leader of this good work in Massachusetts. This is not the place to relate the growth and development of the new department, magnificent as its proportions are, but it is the place to say that Dr. Briggs followed every detail of this work with an earnestness and a vigor that were no small part of its success. He continually suggested new ideas and, not content with merely making the suggestions, saw to it that they were put into effect.

The house at 64 Beacon Street became the center or focal point from which emanated much of the drive behind forward-looking movements for the better care of the mentally sick. The whole base was broadened, so that psychiatry, at last let out of the institutions, became a living force permeating the schools and prisons, as well as the hospitals. With it all a gentleness and kindliness beamed forth so continually that all in trouble — however bad they may have been, however low their origin, however unattractive their cause — had a kind considerate hearing, and were given wholesome advice at 64 Beacon Street. Here persons with a great idea to propose found a ready enthusiasm to support them; here people crushed by adversity also went, and found a tender helping hand ready to direct them to a source of comfort or help.

In the midst of this herculean task of organizing the care of the mentally sick in Massachusetts came World War I. Dr. Briggs was immediately found in uniform. While others were wondering what was the best thing for them to do, he was at Camp Devens putting mental hygiene into the Army. Soon, surrounded by a group of fellow officers, he demonstrated that even the conservatism of the military could not dampen his ardor and determination that the mentally sick should not be taken into the Army, and that if they got there, they should have the best care. Soon the great days in France came about. He seemed to be everywhere. At any rate, he knew everyone of consequence in the A.E.F. and was one of the important figures in the Medical Department of the Army. He became an intimate friend of General Ireland and through this friendship was able to exert considerable influence in matters pertaining to military medicine. He rejoiced in the title of "Colonel" and often used it on his stationery. He often said humorously that the War Department was determined to retire him at the age of ninety.

When the war was over, there was still plenty to do. From early morning until late at night, there was incessant activity. The Division of Mental Hygiene in the Department of Mental Health was to be organized and supported. But the crowning achievement was the passage of the Briggs Law in 1923. For a hundred years there had been provisions for the examination of prisoners accused or suspected of crime, but the initiative rested entirely with partisan lawyers or indifferent officials. Out of the void came the Briggs Law, a provision ensuring the examination of persons accused of major crimes by a nonpartisan board selected by the Department of Mental Health with only nominal remuneration. This law, copied

elsewhere and praised by all, needed support for a while. This support Dr. Briggs valiantly gave. He continually tested it out and sought revision until at last it stands a model for all and a permanent monument to Dr. Briggs.

The need for classifying the inmates of the prisons had long been felt, and yet efforts to bring this about proved quite ineffective until in one interview with Governor Joseph B. Ely, Dr. Briggs succeeded in having a division set up and a budget provided for the examination and classification of inmates in prisons.

Then came the mellower days — still much to do about military affairs, still much concern about the workings of the state hospital system, but interspersed with trips to Europe, visits to the South in the winter, and longer seasons at Hancock in the summer. No one ever sat at table with Dr. Briggs as host, with every morsel of food selected and prepared under his direction, and bounteously served, without coming under the spell of his friendliness and his influence. People came from everywhere seeking advice or information — all sorts of people of all grades of society wended their ways to his fireside, and they were cordially received and directed to the best source of further information. He had a peculiarly canny side to his mind that instinctively separated and discarded the irrelevant and fastened on that part of information which was pertinent. Although his whole life was spent with mental disease, one never heard him speak of hallucinations or delusions, complexes or frustrations. He dealt with people in trouble and knew in round numbers how to help them. Although his life was spent with leaders in every walk, he always championed the cause of the underdog.

There were signs for some time that things were not right with his vascular system. This received due attention, but had no effect whatever on his emotional life. It is fitting and characteristic that in the edition of the Tucson newspaper giving an account of Dr. Briggs's death, under the heading, "Voice of the People," was a stirring letter from him to the Governor of Arizona, ending as follows:

> I have never met Dr. ——, but I have heard a great deal about his skill and intelligence in the care of the mentally ill, and of his knowledge of the requisites for their care and treatment. In forcing Dr. —— to resign because of insufficient appropriations, Arizona cannot but lose the respect of her sister states. This decision to tie the hands of a scientist and an expert should cause those responsible to hang their heads in shame. They have squarely turned their backs on the unfortunate sick of your state.

A. W. S.

MEDICAL EPONYM

Braxton Hicks Maneuver

The maneuver for which John Braxton Hicks (1823–1897), assistant physician-accoucheur at Guy's Hospital, London, is remembered is that described below. The quotation is from a paper "On a New Method of Version in Abnormal Labour," which appeared in the *Lancet* (2: 28–30 and 55, 1860).

> Introduce the left hand, with the usual precautions, into the vagina, so far as to fairly touch the foetal head, even should it recede an inch. . . . Having passed one or two fingers (if only one, let it be the middle finger) within the cervix, and resting them on the head, place the *right* hand on the *left* side of the breech at the fundus uteri. . . . Employ gentle pressure and slight impulsive movements on the fundus towards the right side, and simultaneously on the head towards the left iliac fossa. In a very short time it will be found that the head is rising and at the same time the breech is descending. The shoulder is now felt by the hand in place of the head . . . it in like manner is pushed to the left, and at the same time the breech is depressed to the right iliac fossa. The foetus is now transverse; the knee will be opposite the os, and, the membranes being ruptured, it can be seized . . . and brought into the vagina. . . .
>
> I . . . shall for the present confine myself to the advantages this method gives us in *placenta praevia* — at least, in every form of partial insertion. . . .
>
> . . . as soon as the finger can enter the os, it is practicable to bring down the knee or the foot, and by that means have the command of the haemorrhage and labour at the same time; for the conical form of this natural plug is just suited to the requirements of the case. By gentle traction on the limb, the os is completely and circularly filled up; and as it dilates, the size of the plug, as it descends, increases, whereby a continuous pressure can be kept up, its force varying at will according to the requirements of the case. Having then secured this plug, by keeping hold of the limb, we can afford to wait till, on the one hand, the system has rallied to bear the completion of the labour; or till, on the other, the os has dilated to permit the evacuation of the foetus.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

THE ONE HUNDRED AND SIXTIETH ANNIVERSARY

Wednesday and Thursday, May 21 and 22, The Copley-Plaza, Boston

The exercises of the one hundred and sixtieth annual meeting of the Society will be held at The Copley-Plaza, Boston, on Wednesday and Thursday, May 21 and 22. Members of the medical profession are cordially invited to attend.

The Committee of Arrangements in charge of this year's meeting consists of Drs. Edward J. O'Brien, chairman, William T. O'Halloran, James A. Halsted, George P. Sturgis and Henry H. Faxon.

With the exception of some of the section round-table meetings and luncheons, all the meetings will be held in The Copley-Plaza. The technical exhibits will fill the ballroom and part of the foyer, whereas the scientific exhibits will take the balance of the foyer, the balcony boxes and the east corridor of the main floor. There will be a continuous exhibit of medical motion pictures in the State Dining Room.

The members' registration desk will be in the lobby, and it is hoped that everyone who attends will register.

The annual dinner and the Shattuck Lecture will be held on the same evening, Wednesday, May 21.

The golf tournament will be held at the Commonwealth Country Club on Wednesday, May 21.

Section round-table meetings and luncheons will be held on Wednesday, May 21. As it is necessary to have, beforehand, some idea of the number who will attend these luncheons, all members are urged to notify the committee at once if they plan to come.

WEDNESDAY, MAY 21

General Clinical Meeting

9:15 to 12:00

SHERATON ROOM

Chairman: Dr. Bennett F. Avery.

9:15 *The Normal Needs and the Indications for Vitamin Therapy During Pregnancy.* Dr. Duncan E. Reid, Boston.

9:30 *Vitamins in War.* Robert S. Harris, Ph.D., Cambridge (by invitation)

9:45 *The Clinical Value of Vitamins.* Dr. William H. Sebrell, Jr., Washington, D. C.

10:15 *Enlargement of the Heart.* Dr. Paul D. White, Boston.

INTERMISSION

10:45 *Lupus Erythematosus.* Dr. Richard S. Weiss, St. Louis, Missouri.

11:15 *Alcoholism, a Problem on a Medical Service.* Dr. John A. Foley, Boston.

11:30 *Recent Advances in Chemotherapy of Infectious Diseases.* Dr. Chester S. Keefer, Boston.

11:45 *Some Uses and Abuses of Chemotherapy in Pneumonia.* Dr. Maxwell Finland, Boston.

Supervising Censors' Meeting (PARLOR A) 10:00

Council Meeting (SWISS ROOM) 10:30

Nominating Committee (PARLOR A)

Cotting Luncheon (SWISS ROOM) (following Council Meeting)

Section Round-Table Meetings and Luncheons
12:00 to 2:00

All round-table luncheons will be $1.00. Reservations should be made at once.

Section of Medicine

UNIVERSITY CLUB

Chairman: Dr. Albert A. Hornor, Boston.

Secretary: Dr. Charles L. Short, Boston.

Subject: *The Use and Abuse of Drugs in Heart Disease.* Dr. Paul D. White, Boston.

Section of Obstetrics and Gynecology

UNIVERSITY CLUB

Chairman: Dr. M. Fletcher Eades, Newtonville and Boston.

Secretary: Dr. Raymond S. Titus, Boston.

Subject: *The Management of Bleeding in the Last Three Months of Pregnancy.* Dr. Frederick C. Irving, presiding chairman.

Section of Surgery

STATE SALON, THE COPLEY-PLAZA

Chairman: Dr. R. Nelson Hatt, Springfield.

Secretary: Dr. Stanley J. G. Nowak, Boston.

Subject: *Fluid Administration in Surgery in the Light of Recent Advances in Water and Electrolytic Balance.* Dr. John Scudder, New York City. Discussion by: Dr. Carl W. Walter, Boston. Speaking begins at 12:45 p.m.

Section of Pediatrics

HOTEL LENOX

Chairman: Dr. Harold L. Higgins, Boston.

Secretary: Dr. James M. Baty, Belmont and Brookline.

Subject: *Symposium: The conditioned reflex of Pavlov:*

1. *Physiologic Status.*

2. *Practical Clinical Application, Especially to Children.*
   a. *Gastrointestinal, urinary, respiratory and circulatory tracts.*
   b. *Conditioned behavior.*

Drs. Roy G. Hoskins, George E. Gardner and Harold L. Higgins, Boston.

Section of Dermatology and Syphilology

HOTEL LENOX

Chairman: Dr. William P. Boardman, Boston.

Secretary: Dr. Jacob H. Swartz, Boston.

Subject: *Treatment of Common Skin Diseases.* Dr. Richard S. Weiss, St. Louis, Missouri.

Section of Radiology and Physiotherapy

HOTEL LENOX

Chairman: Dr. Claude L. Payzant, Boston.

Secretary: Dr. Joseph H. Marks, Boston.

Subject: *Problems in the Management of Peripheral Vascular Disease.* Dr. Irving S. Wright, New York City.

Massachusetts Members of The New England Society of Anesthesiology

THE COPLEY-PLAZA

Presiding chairman: Dr. George C. Moore, Boston.

Subject: *Discussion of Anesthesia.*

General Clinical Meeting

2:00 to 5:00

SHERATON ROOM

Chairman: Dr. C. Sidney Burwell

2:00 *The Value of the Roentgen Examination in the Diagnosis of Intestinal Obstruction.* Dr. Eugene P. Pendergrass, Philadelphia.

2:20 *Indications for the Use of the Miller–Abbott Tube.* Dr. William O. Abbott, Philadelphia.

2:50 *Acute Intestinal Obstruction.* Dr. Leland S. McKittrick, Boston.

INTERMISSION

3:30 *Medical Organization.* Dr. Frank H. Lahey, Boston.

4:00 *Conservative Treatment of Occlusive Arterial Disease.* Dr. Irving S. Wright, New York City.

4:30 *Clinical Classification and Diagnosis of Hemorrhagic Diatheses.* Dr. William C. Moloney, Boston.

Annual Dinner (SHERATON ROOM) 7:00

The Shattuck Lecture (SWISS ROOM) 8:45

Thrombosis and Thrombophlebitis. Dr. Alton Ochsner, professor and director, Department of Surgery, Tulane University, New Orleans.

THURSDAY, MAY 22

Symposium on Hormones

9:00 to 10:45

SHERATON ROOM

Chairman: Dr. Charles E. Mongan

9:00 *Complications of Thyroid Substitution Therapy.* Dr. William T. Salter, Boston.

9:15 *The Treatment of Diabetics With Onset in 1941.* Dr. Elliott P. Joslin, Boston.

9:30 *Problems in the Recognition and Treatment of Testicular Insufficiency.* Dr. Allan T. Kenyon, Chicago.

10:00 *The Use of Female Sex Hormones in Disorders of Women.* Dr. George van S. Smith, Brookline.

10:15 *The Current Status of the Female Sex-Hormone Problem.* Dr. Roy G. Hoskins, Boston.

10:30 *The Place of Irradiation in Cancer of the Breast.* Dr. Frederick W. O'Brien, Boston, and Dr. Eugene J. McDonald, Boston (by invitation)

INTERMISSION

Annual Meeting (SHERATON ROOM) 11:00

Annual Discourse (following meeting): *The Role of the Physician in a Competitive Society.* Dr. A. Warren Stearns, Boston.

Annual Luncheon (SWISS ROOM) (following discourse)

Military Symposium

2:00 to 5:00

SHERATON ROOM

Chairman: Dr. Dwight O'Hara, Boston

2:00 *Anesthesia in War Circumstances.* Dr. Wesley Bourne, Montreal.

2:30 *Treatment of Compound Fractures Resulting from Enemy Projectiles.* Dr. Philip D. Wilson, New York City.

3:00 *The Problem of Fatigue in Aviators.* Ross A. McFarland, Ph.D., Boston (by invitation)

INTERMISSION

3:30 *Treatment of Gunshot Wounds of the Brain.* Dr. Gilbert Horrax, Boston.

3:50 *A Report of Newer Work on the Shock Problem.* Dr. Walter B. Cannon, Boston.

4:10 *War Neuroses.* Dr. Douglas A. Thom, Boston.

4:30 *Blood and Blood Substitutes in the Treatment of Hemorrhage, Secondary Shock and Burns.* Dr. Stuart Mudd, Philadelphia.

Motion Pictures

STATE DINING ROOM

Wednesday — May 21

9:30 *The Mechanism and Electrocardiographic Registration of the Heart in Health and Disease* (2 reels).

10:15 *The Complications of the Second Stage of Labor.*

10:30 *Latzko Extraperitoneal Cesarean Section.*

10:45 *The Treatment of Asphyxia Neonatorum.*

11:15 *Epidemic Meningitis of Infancy.*

11:30 *Bronchoscopy and Esophagoscopy on the Cadaver and the Living.*

11:45 *Peritoneoscopy.*

2:00 *Treatment of Burns.*

2:30 *First Aid in Transporting Fractures of the Leg, Spine and Arm.*

2:45 *Technic of Blood Transfusion.*

3:00 *Preparation and Preservation of Pooled Plasma.*

3:15 *The Use of the Miller–Abbott Tube for Decompressing the Small Bowel in Certain Types of Intestinal Obstruction.*

3:45 *Cholecystectomy and Choledochostomy.*

4:00 *Pernicious Anemia.*

Thursday — May 22

9:30 *The Relation of Absorbable Sutures to Wound Healing.*

10:30 *Subtotal Thyroidectomy for Primary Hyperthyroidism.*

10:45 *Diagnosis and Treatment of Infections of the Hand.*

11:30 *Tests of Vestibular Function.*

11:45 *The Administration of Oxygen by Oropharyngeal Catheter.*

2:00 *Myasthenia Gravis: The Erb–Goldflam syndrome.*

2:15 *Vitamin B Complex: Diagnosis, treatment and results of cases of vitamin B deficiency* (sound film).

2:45 *Aftercare of Poliomyelitis.*

3:15 *Technic for Cataract Operations* (colored film).

3:30 *Methods for the Determination of the Bleeding Tendency.*

3:45 *Surgical Treatment for Tuberculoma of the Cecum by Resection with the Aseptic Basting-Stitch Technic.*

4:00 *Hemorrhoids and the Method of Hemorrhoidectomy.*

4:15 *Diagnostic Procedures in Tuberculosis.*

Scientific Exhibits

LOBBY

Booth

A-B *Tuberculosis: Cancer.* Massachusetts Department of Public Health.

FOYER

66-67 *Mycology.* Department of Dermatology, Harvard Medical School and Massachusetts General Hospital. Exhibitors: Drs. Jacob H. Swartz and Ethel M. Rockwood.

68 *Chemotherapy in the Treatment of Infectious Diseases.* Evans Memorial and Massachusetts Memorial Hospitals. Exhibitors: Drs. Chester S. Keefer and Charles H. Rammelkamp.

69 Massachusetts Memorial Hospitals, Department of Surgery.

a. Management of a Blood Bank. Exhibitor: Dr. Frank E. Barton.

b. Experience with the Fenestration Operation in Otosclerosis. Exhibitor: Dr. Leighton F. Johnson.

c. The Use of a Vitallium Tube for Relief of Stricture of the Common Bile Duct. Exhibitor: Dr. Howard M. Clute.

70-72 *Tumor Clinic.* Boston Dispensary. Exhibitor: Dr. Paul R. Hinchey and associates.

BALCONY

1 *Color Photography in Medicine.* Fallon Clinic, Worcester. Exhibitor: Dr. John Fallon.

2 *Broncho-esophagology.* Massachusetts Eye and Ear Infirmary. Exhibitor: Dr. Lyman G. Richards.

3 *Problems in Fractures.* Faulkner Hospital. Exhibitors: Drs. Gordon M. Morrison and Harvey R. Morrison.

4-5 *Clinical Surgery.* The Staff of the Children's Hospital. Exhibitors: Dr. William E. Ladd and associates.

6 *The Visual Presentation of Case Histories for Publication and Teaching.* Massachusetts General Hospital. Exhibitor: Miss Muriel McLatchie.

7 Boston Medical Library.

8 The Lahey Clinic.

a. Surgical Procedures in Complete Colectomy for Intractable Ulcerative Colitis. Exhibitors: Drs. Richard B. Cattell, Neil W. Swinton and Everett D. Kiefer.

b. The Shoulder Joint: Technic of surgical approach. Exhibitors: Drs. Gilbert E. Haggart and James W. Toumey.

c. Hematological Problems. Exhibitor: Dr. John W. Norcross.

EAST CORRIDOR

A-C *Anesthesia.*

a. Advent of Anesthesia. Massachusetts General Hospital.

b. Suction Therapy in the Management and Treatment of Postoperative Pulmonary Complications. Lahey Clinic. Drs. Urban H. Eversole and Carlton R. Souders.

c. Tovell's Technic for Parenteral Fluids. Beverly Hospital.

d. Oxygen Therapy. Massachusetts Memorial Hospitals.

e. Organization of a Hospital Anesthesia Department. Faulkner Hospital.

f. Intravenous Anesthesia. Boston City Hospital.

D-E *Myasthenia Gravis: Recent advances in diagnosis and treatment.* Massachusetts General Hospital. Exhibitors: Drs. Henry R. Viets and Robert S. Schwab.

F-G *Surgical Treatment of Arterial Hypertension.* Beth Israel Hospital. Exhibitors: Drs. David Ayman, Archie D. Goldshine and Reginald H. Smithwick.

H *First Aid in Highway and Skiing Accidents.* Massachusetts Regional Committee on Fractures and Trauma, American College of Surgeons (Dr. A. William Reggio, chairman), Boston Metropolitan Chapter, American Red Cross, on Highway First Aid (Dr. A. William Reggio, chairman; Mr. George P. Johnson, director) and National Ski Patrol (Dr. Charles C. Lund and Mr. Robert Livermore, Jr.).

Additional Exhibit

BALLROOM

Massachusetts Association of Occupational Therapy.

Technical Exhibits

BALLROOM AND FOYER

	Booth No.
Abbott Laboratories	49
The Alkalol Company	36
American Hospital Supply Corporation	39
Ernst Bischoff Company, Inc.	9
The Borden Company	37
Brewer & Company, Inc.	13 and 14
Burroughs Wellcome & Co. (U. S. A.) Inc., New York	30
Campbell X-Ray Corp.	20
The Children's House	61
Crosbie–Macdonald	22
The Coca-Cola Company	62 and 63
Davies, Rose & Company, Limited	3
The DeVilbiss Company	24
The Doho Chemical Corporation	18
The Dy-Dee Service	42
J. H. Emerson Company	27
H. G. Fischer & Co.	53
C. B. Fleet Co., Inc.	45
General Electric X-Ray Corporation	7
Gerber Products Company	6
J. E. Hanger, Inc.	11
Hanovia Chemical and Manufacturing Company	26
H. J. Heinz Company	5
Horlick's Malted Milk Corporation	32
Jones Metabolism Equipment Co.	31
Kalak Water Co. of New York, Inc.	10
Kleistone Rubber Co., Inc.	60
Lederle Laboratories, Inc.	43
Lee DeForest Laboratories	23
The Liebel–Flarsheim Co.	8
Eli Lilly and Company	14A
M & R Dietetic Laboratories, Inc.	4
The Macmillan Company	19
E. F. Mahady Company	56
Massachusetts State Pharmaceutical Association	40
McIntosh Electrical Corporation	16
Mead Johnson & Company	1
The Medical Protective Company	48
Mellin's Food Company of North America	41
The Mennen Company	58
The C. V. Mosby Company	50
T. J. Noonan Company	64
Parke, Davis & Company	46
The E. L. Patch Company	17
Pet Milk Sales Corporation	54
Petrolagar Laboratories, Inc.	33
Philip Morris & Co. Ltd., Inc.	12
Picker X-Ray Corporation	52 and 59
Thomas W. Reed Company	38
S. M. A. Corporation	34
Schering Corporation	57
Sharp & Dohme, Inc.	65
Smith, Kline & French Laboratories	29
E. R. Squibb & Sons	15
Standard X-Ray Sales Corporation	25
Frederick Stearns & Company	47
The Sun-Rayed Company	51
Surgeons and Physicians Supply Company	44
Tailby–Nason Company	21
Westinghouse X-Ray Company, Inc.	28
White Laboratories, Inc.	35
Winthrop Chemical Company, Inc.	55
John Wyeth & Brother, Inc.	2

LADIES' PROGRAM

For the ladies in the families of the members of the Massachusetts Medical Society, a most interesting program has been arranged by the Ladies' Committee, consisting of Mrs. Walter G. Phippen, Mrs. William T. O'Halloran, Mrs. Bennett F. Avery, Mrs. Theodore L. Badger, Mrs. Edmund J. Butler, Mrs. William Dameshek, Mrs. David C. Ditmore, Mrs. John G. Downing, Miss Mary L. Field, Mrs. Reginald Fitz, Mrs. John A. Foley, Mrs. Arthur J. Gorman, Mrs. Thomas H. Lanman, Mrs. Roger I. Lee, Mrs. Frederick J. Lynch, Mrs. Wilfrid C. Macdonald, Mrs. Frank W. Marlow, Jr., Mrs. Donald Munro, Mrs. Robert N. Nye, Mrs. Frank R. Ober, Mrs. Eugene E. O'Neil, Mrs. John W. Spellman, Mrs. Martin H. Spellman, Mrs. A. Warren Stearns, Mrs. Augustus Thorndike, Jr., and Mrs. Charles F. Wilinsky.

WEDNESDAY, MAY 21

All ladies should register at the desk in the main lobby of The Copley-Plaza. In the afternoon there will be a visit to the Isabella Stewart Gardner Museum, Fenway Court, including a tour of the museum, music, a reception and tea; admission to the museum is $0.75. Busses will leave The Copley-Plaza at 2:15 p.m., and there will be no charge for transportation. Those wishing to play golf on Wednesday should make arrangements at the registration desk.

Dinner will be held at the Ritz Roof, Hotel Ritz-Carlton, at 7:30 p.m. Mrs. Phippen, Mrs. Lee and the wives of the district presidents will meet the guests. During and after dinner there will be a floor show. The cost of the dinner tickets is $2.50.

THURSDAY, MAY 22

The registration desk will be open from 9:00 a.m. to 12:00 m. At 10:00 a.m. busses will leave The Copley-Plaza on a most interesting trip to the South Shore. Stops will be made at the Dorothy Quincy House and the Adams Mansion, which was the home of two United States presidents, in Quincy. At Hingham, the Cushing Homestead, which was built in 1686, will be visited by invitation, and a stop will be made at the historic Old Ship Church. Luncheon will be served at Hugo's in Scituate at 1:30 p.m., for which a charge of $1.00 will be made. The return trip will be made by way of Jerusalem Road, Cohasset. There will be no charge for transportation on this trip.

ANNUAL GOLF TOURNAMENT

The annual golf tournament of the Massachusetts Medical Society will be held at the Commonwealth Country Club on the afternoon of Wednesday, May 21.

The Burrage Bowl, emblematic of the Society championship, will again be in competition. Numerous other prizes will be offered for both net and gross scores.

Play will commence at 1:00 p.m., and the greens fee for eighteen holes will be $2.00. If two or more rounds are played during the afternoon the fee will be $2.50. State handicaps will be used.

Luncheon may be had at the club, and all the privileges of the club will be open to the members of the Society who enter the tournament. For additional information, telephone KENmore 2094 or a member of the Committee of Arrangements.

COMMITTEE ON POSTGRADUATE INSTRUCTION

There will be a luncheon and meeting of the district chairmen of the Committee on Postgraduate Instruction at The Copley-Plaza on Thursday, May 22, at 12:30.

MASSACHUSETTS MEDICO-LEGAL SOCIETY

There will be a meeting of the Massachusetts Medico-Legal Society in the State Salon at The Copley-Plaza on Wednesday afternoon, May 21, at 3:00.

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

TOXEMIA OF PREGNANCY, ASSOCIATED WITH ECLAMPSIA AND RESULTING IN DEATH

A twenty-year-old primipara had been hospitalized in a state institution during the last four months of her pregnancy. Because of toxemia, labor was induced.

The family history and past history were not available. The patient was given adequate prenatal care after the fifth month, the urine and blood pressure being checked twice a week. Albumin did not appear until the thirty-second week, and edema appeared at the thirty-fourth week. At the twenty-second week, a blood pressure of 130 systolic, 75 diastolic, was recorded. The last week or so before induction of labor, the blood pressure was said to have gone up from 130 to 180 systolic.

Because the patient's condition was not improving, induction of labor was attempted. A catheter was inserted in the uterus and left in place for ten hours. Then, because labor had not begun, the membranes were ruptured and the cervix was packed. Six hours later, labor began. Thirty-six hours after the catheter was inserted, the patient had a severe convulsion and became unconscious. At that time the cervix was dilated to admit three fingers, and delivery was accomplished by manually dilating the cervix and applying high forceps. The fetus was dead. Ten minutes after the birth of the placenta, which was said to have been normal, the patient had a circulatory collapse and died an hour and a half later.

Comment. It is a general concept that a toxemia culminating in eclampsia and developing in a patient under constant observation during the last five months of pregnancy means only that the condition was not intelligently or adequately treated. There is no evidence in this case that there was any pelvic disproportion. Had there been any, cesarean section would have been the operation of choice. It is also possible that if there had been an appreciable rise in blood pressure occurring within twelve to twenty-four hours and a diminution of the urinary output, cesarean section again would have been indicated. Had one decided that induction was the best method of treatment, rupturing the membranes, treating the patient conservatively by the use of magnesium sulfate or veratrum viride to control the blood pressure and leaving the case absolutely alone after the membranes had been ruptured might well have resulted in a successful outcome. *Accouchement forcé* and high forceps are procedures that the unconscious eclamptic patient stands very poorly. Extreme vaginal conservatism should always be the rule.

Just what caused the circulatory collapse is hard to determine. It may well have been that the heart collapsed, either from shock or from the toxemia of the eclampsia; it is barely possible that the patient had a cerebral hemorrhage. These, however, are not very common. Unfortunately no autopsy was performed.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

DEATHS

DEWEY — CHARLES G. DEWEY, M.D., of Dorchester, died April 18. He was in his eighty-second year.

Born in Hanover, New Hampshire, he attended Dartmouth College and received his degree from the Dartmouth Medical School in 1886. For four years he was assistant superintendent at the Boston City Hospital. From 1901 to 1934 he was examining physician of the mentally ill for the Institutions Department of the City of Boston, and he was associate commissioner of the Massachusetts Department of Mental Diseases from 1916 to 1934.

He was a member of the Massachusetts Medical Society, the American Medical Association and the American Psychiatric Association.

His widow, a son and two daughters survive him.

MELLEN — ELEANOR W. MELLEN, M.D., of Newton Highlands, died April 19. She was in her seventy-third year.

Dr. Mellen received her degree from the Woman's Medical College of Pennsylvania in 1903, and was a member of the Massachusetts Medical Society and the American Medical Association.

MURRAY — MICHAEL E. MURRAY, JR., M.D., of Cambridge, died April 21. He was in his thirty-seventh year.

Born in Menominee, Michigan, he attended Notre Dame and received his degree from the Harvard Medical School in 1932. He was on the staff of the Massachusetts General Hospital, and was an assistant in the Hygiene Department of Harvard University.

Dr. Murray was a member of the Massachusetts Medical Society and the American Medical Association.

His widow and two small daughters survive him.

MISCELLANY

CITY HEALTH CONTEST AWARDS

Ten cities were named as 1940 winners of the Inter-Chamber City Health Conservation Contest conducted annually by the United States Chamber of Commerce in co-operation with the American Public Health Association; these include Newton, Massachusetts, and Hartford and Greenwich, Connecticut. Awards were also given in 1940 for the most effective programs for the control of tuberculosis and syphilis, the contest being sponsored by the Metropolitan Life Insurance Company; the two winners in the first group were Newton, Massachusetts, and Hartford, Connecticut.

AMERICAN MEDICAL GOLFING ASSOCIATION

The American Medical Golfing Association will hold its twenty-seventh annual tournament at the Cleveland Country Club and Pepper Pike Club in Cleveland, Ohio, on Monday, June 2. The two clubs have courses of championship caliber, and the clubhouse of the Cleveland Country Club is one of the most beautiful and spacious in the country.

More than 250 members of the association are expected to take part in the thirty-six-hole competition. The hours for teeing off are from 7:30 a.m. to 2:00 p.m. The fifty prizes in the eight events will be distributed after the banquet, which will be held at the clubhouse of the Cleveland Country Club at 7:00 p.m.

All male members of the American Medical Association are eligible and cordially invited to become members of the association; applications may be obtained from the executive secretary, Bill Burns, 2020 Olds Tower, Lansing, Michigan. Each participant in the tournament is required to present a card with his home-club handicap, signed by the club secretary, at the first tee on the day of play. No handicap over 30 is allowed. Only active members of the association may compete for prizes. No trophy is awarded to a member who is absent from the annual dinner, which is always worth waiting for.

WOMAN'S AUXILIARY, AMERICAN MEDICAL ASSOCIATION

On June 2 to 6 the Woman's Auxiliary to the American Medical Association will hold its nineteenth annual convention at Cleveland. Hotel Carter will be the headquarters. The wives of all physicians who are members of the American Medical Association and their friends are invited to attend the meetings. Requests for reservations should be sent to Dr. Edward F. Kieger, Committee on Hotels and Housing, 1604 Terminal Tower Building, Cleveland.

VINCENT MEMORIAL HOSPITAL

Plans have recently been announced for the affiliation of the Vincent Memorial Hospital and the Massachusetts General Hospital. The present building of the Vincent Memorial Hospital on South Huntington Avenue, Boston, will be sold, and a new building, still called the Vincent Memorial Hospital, will be built on the grounds of the Massachusetts General Hospital. Dr. Joe V. Meigs, chief-of-staff at the Vincent Memorial Hospital, and the other physicians on the staff will continue to serve, and Dr. Meigs will be made head of a newly established gynecological unit at the Massachusetts General Hospital. At the meeting of the Vincent Club — an effective supporter of the Vincent Memorial Hospital — at which this announcement was made, Mr. Charles Francis Adams said that the affiliation was "a step forward in efficiency, a step forward in good management and a step forward in service to the community."

NOTES

Dr. Henry K. Beecher, associate in anesthesia, Harvard Medical School, has been appointed to a professorship at the Medical School to fill the Dr. Henry Isaiah Dorr Chair of Research and Teaching in Anaesthetics and Anaesthesia.

Dr. Israel Kapnick, '38, of Providence, Rhode Island, has been awarded the Louis E. Kirstein Fellowship at the Harvard Medical School for the summer of 1941. The fellowship, to promote "scientific medical education," was established through a gift of $28,550 made to Harvard University by eighty-nine friends of Mr. Kirstein.

CORRESPONDENCE

TUBERCULOSIS AND GENIUS

To the Editor: In the March 6 issue of the *Journal* there appeared a review of the book, *Tuberculosis and Genius*, by Dr. Lewis J. Moorman. That review, in my opinion, not only was unkind and unfair, but also did grave injustice to Dr. Moorman and his work.

About half the space given to the book review was occupied by a harangue about the various abuses of present-day management of tuberculosis sanatoriums,

which was not only unwarranted in the light of facts, but was entirely missing the mark so far as Dr. Moorman's book is concerned, since none of the characters considered in the biographical sketches came under strictly modern management. I shall not say anything here about certain unfortunate expressions used by the reviewer, such as "victims of tuberculosis . . . herded together," "forced to submit to the tyrannical rules of community life" and "abnormal conditions imposed upon them." Who has ever relegated tuberculous patients among the morons or exalted them to the rank of geniuses? Who has branded them as dissolute erotics or beatified them as angels of chastity? It is true that a London physician, Dr. Gwilym, once proposed castration as a method of treatment for pulmonary tuberculosis. But that was in 1857. Strong objections being raised, he desisted. As to clinicians who feel "the urge to become amateur psychiatrists," I think that chest specialists are perfectly justified in studying their patients as best they can in that respect and in noting carefully their *emotions*, *reactions* and *adjustments* during the course of their disease. All that, however, has nothing to do with Dr. Moorman's book.

Are the diagnoses made by Dr. Moorman correct? Is there a relation between tuberculosis and genius? And, if there is, what is the nature of that relation? Those are the questions raised by Dr. Moorman's essays.

To make a retrospective diagnosis on a person dead long ago is not any different than to study any other point of history, and the two should be governed by the same rules. All the records extant should be carefully and critically examined before conclusions are drawn. Now, everyone familiar with the study of history knows that, even when all the evidence has been gathered, it is very often impossible to arrive at deductions that are certain, positive and final. One must often content oneself with conclusions that are *most probable*, or only *probable*, or simply *possible*. The same holds true of a diagnosis. If I must show that a man has had a positive sputum before I can call him tuberculous, then none of Dr. Moorman's characters had tuberculosis. Nobody had tuberculosis before Koch discovered the bacillus. Did Stevenson have it? I must doubt it even if all the rest of the evidence points toward it; because, if I am not mistaken, Trudeau failed to examine Stevenson's sputum when he took the latter to his laboratory.

The fact that all the diagnoses made by Dr. Moorman can be questioned is not Dr. Moorman's fault. It is the fault of history. History is not an exact science, like mathematics, and never will be. It deals with human beings, and its conclusions are never definitive and can always be questioned. For example, historians all over the world have not arrived yet at a positive and certain conclusion about the true cause of World War I. Likewise, the man interested in medicohistorical studies has to be satisfied with diagnoses that are sometimes only probable, sometimes only possible.

The reviewer contends that Dr. Moorman's excursions into history and his diagnoses are based on assumptions. Not at all. They are based on the available records. Has Dr. Moorman examined *all* the available records? That is what should have been discussed. The book should have been studied like a book of history, with the methods of the historian. The reviewer has failed to do that. More than that, after claiming that Dr. Moorman walks on very thin ice, he himself goes out on thinner ice, and proposes, for Voltaire, a diagnosis of bronchiectasis. There are at least thirty different medicohistorical studies about Voltaire, his health, his diseases and his last illness. If there is a diagnosis that is *most improbable*, it is that of bronchiectasis.

What is the relation between tuberculosis and genius? That question calls for another and more inclusive one. What are the relations between the body and the mind? Genius is only the mind working in a superlative way. It is a truism to say that the body and the mind are closely interrelated and that states of mind are often dependent on states of body and, conversely, that bodily changes often follow mental changes. It is not the place here to inquire into the modifications wrought on the mind and the personality by changes in the secretions of the endocrine glands, by chronic infections, by intoxications of long duration and by other factors. But let us take one example: the thyroid gland. We all know the mental changes that take place when the secretions of this gland begin to decrease: slowness, dullness, apathy. If the function of the gland continues to slow up, we may have actual cretinism. The reviewer says that "genius transcends contingencies." If suffering from the most disabling form of tuberculosis for years is a contingency, then having a thyroid gland that does not secrete is also a contingency. I should like to see a genius transcend that one!

Let us take another example. A composer of talent, or of genius, is occupied in writing a symphony. In the course of the creation of that symphony, a virulent, destructive tuberculous process invades one of his lungs and destroys it. Who will say that the "style" of his music will not be affected by what is going on in his chest?

Dr. Moorman never claimed that tuberculosis is the cause of genius, or gives genius to a man, or adds to the genius of a man. Is there anything more evident and obvious than the fact that having tuberculosis does not presuppose having genius? I feel a little sympathy for Dr. Moorman. I myself, some time ago, in an article about a man who had had tuberculosis, asked, merely asked, if there was not some relation between tuberculosis and the creative mind. Another book reviewer and his prompter tried to have me say that tuberculosis was a causative factor in the production of genius.

The reviewer denies that there is any relation between tuberculosis and genius, but he is willing to admit that the "artistic temperament" can be influenced by syphilis. He does not demonstrate it, however. When he has succeeded in demonstrating that syphilis can influence an artist, he will have demonstrated that tuberculosis can also influence an artist. The reviewer shows confusion on another point. I put side by side two sentences from his review. "Knowing the artistic temperament, one may be permitted to suggest the existence of a sprinkling of spirochetes." And, "To tell them that the creative power of genius is enhanced by the toxins of such or such a germ would seem to be unjustifiable." Isn't there contradiction here?

As to Balzac, DeQuincey and Poe having written beautifully without coffee, opium and alcohol, that is not the question. The question is, Would they have written what they wrote as they wrote it, without coffee, opium and alcohol? A man of genius is born with his genius. The manifestation of his genius is conditioned and determined by several external factors, among which are the development of his body, his health, his diseases and so forth.

To conclude, I think that Dr. Moorman should be congratulated. A man who has had long experience in tuberculosis work, he has written a most interesting book about a certain aspect of tuberculosis. It is not a work of eru-

dition or research. It was not intended to be one. It is a captivating study of the lives of ten men and women handicapped by a long debilitating illness. In the words of Dr. Max Pinner, who has written, in my opinion, the best and most sympathetic review of Dr. Moorman's book, it should "reach many patients and many physicians . . . it will teach physicians a deeper sympathy and understanding for their charges . . . it belongs in the patients' library of every tuberculosis ward." The National Tuberculosis Association has recognized the value of the book and has selected it for distribution among its members.

GABRIEL NADEAU, M.D.

Rutland State Sanatorium,
Rutland, Massachusetts.

PHARMACEUTICAL ADVERTISING

To the Editor: The editorial in the March 13 issue of the *Journal* that deals with vitamins is very interesting, since it tells the medical practitioner that he has given over, perhaps by force, his therapy to the purveyors of proprietary preparations — in this case, vitamins.

Every line in the editorial is full of truth, and if the average physician is anxious to keep away from the poorhouse where he is gradually being forced by many unethical so-called "ethical" houses, he should do what I have been doing in the last seven years.

I do not prescribe anything manufactured or distributed by a concern that advertises its products in a publication that is read by the general public, since it is my belief that such an act on the part of the manufacturer or distributor constitutes the illegal practice of medicine and is thus an interference with the livelihood of the legitimate practitioner. There are a few manufacturers who do not advertise to the general public, nor do they furnish display signs for the use of counter-prescribing druggists. It is therefore the duty of the average physician to confine his prescribing to articles of these manufacturers. By doing this the physician will be able to preserve his own practice and will at the same time aid those who are helping him.

A. PRAGLIN, M.D.

26 Davis Street,
Boston.

REPORTS OF MEETINGS

HARVARD MEDICAL SOCIETY

A regular meeting of the Harvard Medical Society was held at the Peter Bent Brigham Hospital on January 14, 1941, with Dr. Joseph C. Aub presiding.

The first case was that of a forty-one-year-old man who had entered the hospital three months previously because of epigastric and left-upper-quadrant distress. A gastrointestinal series revealed a carcinoma of the upper end of the stomach, and total resection was subsequently carried out, with smooth convalescence. Roentgenographic studies eleven days postoperatively revealed a well-functioning stoma; when the patient was discharged he was asymptomatic. A month later the patient was bothered by heartburn, for which dilute hydrochloric acid and antacid powders were administered, with relief in a week. A recurrence of these symptoms in another ten days was not amenable to this therapy, and the patient also began to be bothered by regurgitation, particularly of saliva. He voluntarily started on a soft-solid diet without relief but with the loss of 15 pounds in weight until the present admission. Physical examination, the blood chemical findings and a gastrointestinal series were negative except for some abnormality of swallowing during the last test. This was not true regurgitation but largely difficulty in swallowing saliva. Atropine in doses of 0.6 mg. daily caused a decrease in the amount of saliva, and 10 units of insulin three times daily improved his failing appetite. No sympathetic symptoms were present, nor was the salivation associated with nausea.

Dr. E. S. Emery suggested that radical gastric surgery may often be associated with autonomic symptoms resulting from an accentuation in a susceptible person of an underlying autonomic imbalance. These manifestations may take the form of sweating, dizziness or an elevation of blood pressure after meals. Dr. Robert Zollinger stated that the much-discussed problem of anemia seldom develops, whereas diarrhea or obstruction due to a poorly functioning stoma is quite frequent. This was the first case of salivation, and one other patient experienced postprandial rhinitis. Dr. Arturo Rosenblueth expressed surprise that atropine did not have a better effect. He stated that abnormal afferent impulses might explain the phenomena. The rate or even presence of regrowth of afferent fibers following gastric resection is an unknown quantity.

The medical case was that of a fifty-nine-year-old man who entered the hospital because of weakness and unsteadiness of his legs for several weeks. Several years previously he had had gonococcal urethritis, which had been "cured" by local treatment. One year before entry weakness and lameness of the lower extremities were first noted. Five weeks before entry he realized that the heat of a stove was not felt with his left hand, and he began to have poor control of finer movements and speech. Blood and cerebrospinal-fluid Wassermann tests were positive at an outlying hospital. Neurologic examination on entry revealed hyperactive tendon reflexes, a positive Hoffmann sign bilaterally, and poor position sense and heel-to-shin test. There was an elevated sedimentation rate. The cerebrospinal fluid revealed a positive Wassermann reaction and a first-zone gold-sol curve. The blood Wassermann reaction was negative three times and positive twice. Bismuth and neoarsphenamine therapy was instituted. Dr. John Romano suggested that the unusual combination of signs was due to a lateral medullary thrombosis superimposed on a diffuse syphilitic meningoencephalitis.

The speaker of the evening was Dr. Rosenblueth, his subject being "A Physiologic Analysis of Abnormal Neuromuscular Function." Dr. Rosenblueth defined neuromuscular disorders as a "deficiency in the performance of motor function due to a lesion below the motor neurone." He stated that the purpose of his address was an attempt to classify such disorders on a physiologic rather than the usual morphologic basis, and to propose some tests for the approval of the clinician.

As an introduction, the speaker showed a schematic plan of the neuromuscular response in which a nerve impulse sets free a quantum of acetylcholine, which causes a muscle impulse (similar in nature to a nerve impulse); this, in turn, results in a contractile response, which is the clinical but not the physiologic end point. The latter involves the inactivation of acetylcholine by cholinesterase, which is found at the site of release and causes a breakdown into acetic acid, inactive choline and a recovery process. It was pointed out that an identical response can be brought about in what is termed a "contracture" when acetylcholine exerts its effect directly without a muscle impulse.

The mechanism whereby acetylcholine causes a muscle

impulse was then discussed. In any nerve-muscle preparation, there is a threshold below which acetylcholine produces no response, but there is also an upper limit to the effective range above which this substance becomes paralytic. Usually there is only a single response to a given amount of acetylcholine because cholinesterase causes so rapid a breakdown that the level of mediator is subthreshold by the time the muscle ceases to become refractory. The failure of a muscle to respond in any case may be due to an elevated threshold in the presence of a normal amount of acetylcholine (curare), or to a diminished amount of acetylcholine in cases in which the threshold remains essentially normal (certain types of fatigue). In Wallerian degeneration the transmission of nerve impulses proceeds for some time before suddenly ceasing. Here the nerve impulses remain normal, but the acetylcholine output eventually falls below the threshold value and responses cease.

The paralytic range of acetylcholine can be demonstrated experimentally by neutralizing cholinesterase with eserine. In this case there is no breakdown of acetylcholine, and a summation of the substance results from repeated stimulations. Therefore, a curare and eserine may achieve the same result by entirely different mechanisms, and it becomes clinically important in a given case of neuromuscular dysfunction to know which type of paralysis is present, for adding acetylcholine aids the first condition but merely aggravates the second.

Dr. Rosenblueth then discussed so-called "fatigue," a poorly defined physiologic as well as clinical term. High-rate stimulation (60 per second) of a nerve-muscle preparation results in fatigue of transmission because of too little acetylcholine. Stimulation at a slow rate (2 to 4 per second), on the other hand, causes contractile fatigue. Here the response, no matter how far diminished, never entirely disappears, in contrast to the previous type of fatigue. Transmission is intact, but there is a depression of the contractile system.

A classification of clinical neuromuscular disorders is suggested on this physiologic basis. Under "hypodynamic" effects would be classified atonia, ataxia, fatigue and paralysis. These might be due to transmission defects in which acetylcholine is either below the threshold level or above the paralytic level, or they might result from an impairment of the motor system in the muscle itself. On the opposite side would be the "hyperdynamic" conditions such as convulsions, spasm, myoclonia and myotonia. Such conditions might be due to nerve changes such as can be caused by "veratrine" when the responses of nerve become repetitive; changes in transmission (possible but improbable), when the action of acetylcholine may be prolonged within the effective range; and dysfunction of the contractile mechanism whereby the response to a given stimulus is greater than normal.

Finally Dr. Rosenblueth suggested certain clinical tests that he considered applicable to neuromuscular disorders to determine the site and type of pathologic physiology: to decide whether a disease is central or peripheral, the carrying out of novocain block and the stimulation of the nerve at various frequencies peripherally to the block; the study of the acetylcholine thresholds of normal and neurologic patients; the administration of eserine to determine whether there is improvement or aggravation of symptoms and signs; the use of subparalytic doses of curare; biopsy and cholinesterase-content determinations of muscle and of blood — these may be difficult to interpret, owing to the heterogeneous distribution of the enzyme in muscle.

The discussion was inaugurated by Dr. John Romano, whose only question concerned the variable effects of quinine obtained in myotonic conditions and the physiologic basis for these effects. Dr. Rosenblueth replied that this drug is now known to be similar to curare in certain respects and to eserine in others. Therefore, complicated reactions may ensue, and the end-results depend on the original balance in the neuromuscular system.

Dr. Henry R. Viets, acknowledging the desirability for improved clinical tests in neuromuscular disorders, advanced certain practical objections to the speaker's suggested tests. In the first place, there are too few peripheral nerves available for stimulation. Cholinesterase is difficult to detect and quantitate in the situations in which it is desirable. It is impractical to treat with acetylcholine, owing to its ready destruction in the body. In regard to treatment of some of these disorders, Dr. Viets stated that prostigmine and other anticurarelike drugs seem to be all right clinically. In Thomson's disease there appears in most cases to be improvement with quinine and aggravation of the clinical picture with prostigmine. The important relation of endocrinology to the entire group of neuromuscular disorders was emphasized, and Dr. Viets reminded the audience that the first description of myasthenia gravis recommended pregnancy as a cure.

In reply, Dr. Rosenblueth suggested that the easily accessible ulnar nerve be used for stimulation on the basis that there is probably generalized neuromuscular involvement, even in such seemingly localized conditions as myasthenia gravis and scapulohumeral muscular atrophy. Dr. Viets agreed with the probability of generalized subclinical involvement and the feasibility of ulnar stimulation.

Dr. William T. Greene suggested that the neuromuscular mechanism in the clinic is not so simple as in the physiology laboratory and that secondary responses often overshadow or complicate primary reactions, particularly when the central nervous system is involved. Dr. Aub asked how often plumbism with poor re-creation of phosphocreatine may cause neuromuscular dysfunction. Dr. Rosenblueth replied that this probably is not rare.

JEWISH MEMORIAL HOSPITAL

A regular meeting was held at the Jewish Memorial Hospital on February 27, with Dr. Samuel A. Levine discussing "Common Errors in the Diagnosis of Cardiac Conditions." It was pointed out that heart disease is one condition in which the diagnosis is almost all-important, for the treatment is usually easy or at least easy to determine. Furthermore, the treatment of heart disease is generally good, especially in large medical centers, but this is not so of diagnosis. An important and common error is the diagnosing of heart disease when none is actually present, for the patient may thus be condemned to a life of inactivity and the use of powerful drugs. To make a positive diagnosis of organic heart disease, which is not necessarily the same as heart failure, one should rely on the three cardinal signs: a diastolic murmur, cardiac enlargement and gallop rhythm. A diastolic murmur invariably means heart disease until proved otherwise, but the observer must be able to distinguish what is diastolic. Although enlargement usually indicates organic heart disease, it may occasionally be reversible and reparable, as in beriberi. Gallop rhythm, except for the rare normal systolic type, is an indication of serious cardiac damage. Other findings are usually present in heart disease but do not necessarily incriminate the organ, whereas a significant number of cardiac patients do not have any of these findings. In angina pectoris the three cardinal signs may well be absent. Here

the symptoms are important, and an accurate history is the *sine qua non*. In the absence of characteristic history all accessory diagnostic aids may need to be employed, such as studies of the pulmonary and biliary systems. Dr. Levine emphasized that apical pain is usually not coronary in origin.

If heart failure is present, the kind and amount of heart disease is not important when the treatment is the same. But one should keep looking for those rare conditions that can be cured and in which the treatment differs from the usual regimen of digitalis and diuretics. The classic examples of this are the masked thyrocardiacs for the proper diagnosis can rehabilitate a patient who might otherwise be considered hopeless. Although such people may on the surface appear perfectly normal, certain important clues indicate the true etiologic factor underlying the cardiac condition. The skin may be moist, pigmented and hyperemic; the heartbeat may be hyperactive, snappy and agile, compared to the thrusting impulse of other cardiac enlargement; there may be signs of thyrotoxicosis, such as weight loss with a good appetite, an elevated metabolic rate and a diminished cholesterol level. Dr. Levine warned that heart failure alone may give an elevated metabolic rate. An increase in the velocity of blood flow as measured by the circulation time clinches the diagnosis in the presence of congestive failure, which in all other conditions decreases the rate.

One of the most important factors in establishing a proper basis for cardiac therapy is the intelligent interpretation of signs and symptoms. Heart failure rather than heart disease should be treated, for organic disease is usually present about five times as long as failure, and early therapy is of no avail. One of the important manifestations of the failing heart is dyspnea. Here one must distinguish the shortness of breath due to obesity, bronchitis, anemia, asthenia and neurotic overventilation. Cardiac treatment does not help these conditions, and in any case should be omitted if there is not symptomatic relief. An obese person may later develop heart disease with dyspnea, but this does not indicate that previous dyspnea was cardiac in origin. Important aids in this differential diagnosis are the vital capacity and the circulation time. Dyspnea with a normal vital capacity is not heart disease. The circulation time is increased in cardiac conditions over those of pulmonary origin. Naturally, roentgenograms are of inestimable value.

A second sign of failure is rales, which should be moist and basal in comparison with the rhonchi and crepitant rales of pulmonary disease. Anterior and expiratory rales usually indicate respiratory disease. Pulmonary carcinoma may cause both dyspnea and rales, and although the proper diagnosis may not always be made, one can reassure one's consultant that the condition is not cardiac.

Edema, a third sign of failure, may be caused by increased pelvic pressure with varicose veins, nephritis and low total-protein values. The last condition may confuse the issue by being the sole cause of edema in a known cardiac patient. Since the blood volume is increased in all cardiac congestions, this may help in differentiating the edema of nephritis.

Finally, the enlarged liver of cardiac failure must be distinguished from carcinoma and cirrhosis, either of which may well occur in the presence of such conditions as hypertension. The elevation of venous pressure found in cardiac conditions is often a helpful differential point.

Dr. Levine cited additional helpful clues in the diagnosis of cardiac conditions. Women do not have coronary disease without hypertension except rarely later in life, but men may. Anginal pain is usually substernal but not lengthy or usually present in recumbency, in contrast to aneurysm. Left-axis deviation is a common finding in patients over forty years of age, especially when there is a high diaphragm, and should therefore not be considered significant in itself. It was stated that the electrocardiogram may be helpful in diagnosis but should not be used for prognosis in the individual case. This is borne out by the fact that although an inverted T wave in Lead 1 usually indicates a bad prognosis, there may be occasional exceptions. Emphysema should always be considered, since it is a common cause of exertional dyspnea, rales and distant heart sounds. If rales are unilateral, they are usually at the right base in cardiac conditions.

Finally, Dr. Levine re-emphasized the importance of searching for those reversible conditions that may be cured, for although each type may be rare, the total number of cases may be appreciable. Remedial arrhythmias alone may cause heart failure in a small group of patients. Arteriovenous aneurysms anywhere in the body may cause eventual cardiac failure by overwork even in an originally normal heart. These are often traumatic in origin, and are accompanied by a steady machinery murmur. Surgical results in such cases are usually strikingly effective. Then there is the beriberi heart, which is so effectively cured by appropriate vitamin therapy. And surgery may bring about a cure in constrictive pericarditis. The three questions that should be asked in any case, therefore, are: Is there heart disease? Is there heart failure? Is there heart failure for which specific therapy is available?

NOTICES

ANNOUNCEMENT

Dr. A. W. Gasson announces the removal of his office from 937 Washington Street, Norwood, to 34 North Main Street, Mansfield.

BOSTON LYING-IN HOSPITAL

There will be a meeting of the Journal Club of the Boston Lying-in Hospital on Wednesday, May 7, at 8:15 p.m. in the lecture room of the hospital. Dr. Alan F. Guttmacher, associate professor of obstetrics, Johns Hopkins University School of Medicine, will speak on "Multiple Pregnancy: Certain historical, biological and clinical aspects."

Physicians and medical students are cordially invited to attend.

HARVARD MEDICAL ALUMNI ASSOCIATION

The annual meeting and dinner of the Harvard Medical Alumni Association will take place on Wednesday, June 4, at 7 p.m. at the Hermit Club, Cleveland, during the annual session of the American Medical Association. The speakers will be Dean C. Sidney Burwell, Dr. Frank H. Lahey, Dr. Roger I. Lee and Dr. Philip D. Wilson.

ANNUAL CONCERT OF THE BOSTON DOCTORS' SYMPHONY ORCHESTRA

The Boston Doctors' Symphony Orchestra will give its second annual concert on Sunday, May 11, at 8:15 p.m. in Jordan Hall. Alexander Thiede will conduct. Dr. Werner Mueller will appear as soloist.

Proceeds of the concert will be used for the establishment of a fund for a free bed in each of the following

hospitals: Beth Israel Hospital, Children's Hospital, Massachusetts Eye and Ear Infirmary and Boston Dispensary. Tickets at $1.00 may be obtained by applying to Dr. Julius Loman, Pelham Hall Hotel, Brookline (BEA 2430).

NORFOLK DISTRICT MEDICAL SOCIETY

The ninety-first annual meeting of the Norfolk District Medical Society will take place on Tuesday, May 6, at the Hotel Puritan, Boston.

PROGRAM

6 p.m. Business meeting.
7 p.m. Dinner.
Colonel Charles W. Furlong, explorer, lecturer and writer, will speak.

Reservations for the dinner should be made by Saturday noon, May 3, and should be sent to Dr. Frank S. Cruickshank, secretary, 1247 Beacon Street, Brookline.

WACHUSETT MEDICAL IMPROVEMENT SOCIETY

The next meeting of the Wachusett Medical Improvement Society will be held at the Holden District Hospital on Wednesday, May 7, at 6:30 p.m.

PROGRAM

Clinicopathological conference.
A medical case. Drs. I. L. Cutler, J. J. Dumphy and A. R. Crane.
A surgical case. Drs. John Fallon and William Freeman.

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

The sixth annual convention of the National Gastroenterological Association will be held at Hotel Commodore, New York City, May 13 to 16. May 15 has been designated New England Day, and many of the speakers will be from this district. Dr. Frank H. Lahey will address the meeting in the afternoon and will be the speaker at the banquet that night. Other New England speakers include Drs. Carl Bearse, Louis F. Curran, Herbert G. Dunphy, E. Stanley Emery, Jr., Charles A. Lamb, Charles W. McClure, William R. Morrison, George Papen, Richard H. Sweet, and Norman A. Welch, of Boston, Drs. Frank A. Cummings, Frederic V. Hussey and E. Ashley Shaw, of Rhode Island, and Dr. Robert F. Scholl, of Connecticut.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING SUNDAY, MAY 4

MONDAY, MAY 5

12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

TUESDAY, MAY 6

*9-10 a.m. Pneumoencephalography. Dr. Kurt Goldstein. Joseph H. Pratt Diagnostic Hospital.

12:15–1:15 p.m. Clinicoroentgenologic conference. Peter Bent Brigham Hospital amphitheater.

WEDNESDAY, MAY 7

*9–10 a.m. Urticaria. Dr. E. A. Brown. Joseph H. Pratt Diagnostic Hospital.

*12 m. Clinicopathological conference. Children's Hospital.

*8:15 p.m. Multiple Pregnancy: Certain historical, biological and clinical aspects. Dr. Alan F. Guttmacher. Journal Club. Boston Lying-in Hospital.

THURSDAY, MAY 8

*9–10 a.m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

FRIDAY, MAY 9

*9–10 a.m. Marked Hyperthyroidism. Dr. Oliver Cope. Joseph H. Pratt Diagnostic Hospital.

SATURDAY, MAY 10

*9–10 a.m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*Open to the medical profession.

MAY 2 — Henry Jackson Lecture. Page 752, issue of April 24.

MAY 5–9 — American Association of Industrial Physicians and Surgeons and American Industrial Hygiene Association. Page 484, issue of March 13.

MAY 7 — Wachusett Medical Improvement Society. Notice above.

MAY 8 — Pentucket Association of Physicians. Page 263, issue of August 15.

MAY 11 — Boston Doctors' Symphony Orchestra. Second annual concert. Page 790.

MAY 12–14 — American Association for the Study of Goiter. Page 753, issue of April 24.

MAY 13–16 — National Gastroenterological Association. Notice above.

MAY 14 — New England Obstetrical and Gynecological Society. Page 752, issue of April 24.

MAY 21 — Massachusetts Medico-Legal Society. Page 785.

MAY 21, 22 — Massachusetts Medical Society, Boston. Page 781.

MAY 28–JUNE 2 — American Board of Obstetrics and Gynecology. Page 262, issue of February 6.

MAY 29–31 — Medical Library Association. Page 671, issue of April 10.

MAY 30, 31 — American Heart Association. Hotel Statler, Cleveland.

MAY 30–JUNE 2 — American College of Chest Physicians. Hotel Statler, Cleveland.

JUNE 2 — American Medical Golfing Association. Page 785.

JUNE 2–6 — American Medical Association. Cleveland.

JUNE 2–6 — Woman's Auxiliary, American Medical Association. Hotel Carter, Cleveland.

JUNE 4 — Harvard Medical Alumni Association. Page 790.

JUNE 22–24 — Maine Medical Association. Marshall House, York Harbor, Maine.

OCTOBER 14–17 — American Public Health Association. Page 579, issue of March 27.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH

MAY 7 — Page 708, issue of April 17.

ESSEX SOUTH

MAY 14 — Relation of the Doctor to the Law. Mr. Leland Powers. New Ocean House, Swampscott.

FRANKLIN

MAY 13 — This meeting will be held at 11 a.m. at the Franklin County Hospital, Greenfield.

MIDDLESEX SOUTH

MAY 14 — Page 752, issue of April 24.

NORFOLK

MAY 6 — Notice above.

WORCESTER NORTH

APRIL 23 — Eighty-second annual meeting. Page 709, issue of April 17.

BOOK REVIEWS

Social and Biological Aspects of Mental Disease. By Benjamin Malzberg, Ph.D. 8°, cloth, 360 pp., with 170 tables and 19 graphs. Utica, New York: State Hospitals Press, 1940. $2.50.

In this book the author publishes a series of papers written during the last several years. It is a very valuable contribution to the subject of psychiatry and provides a large mass of basic data for psychiatrists and students of mental disorders. Dr. Malzberg believes that the increase in patients on the books of the New York State

hospitals between 1909 and 1935 indicates an increase in mental disease in that state. The larger number of patients thus revealed might be due to an increasing length of hospital stay or to changes in parole criteria that would increase the number of patients on the books.

The chapter, "Age and Mental Disease," is very interesting and gives a number of graphs presenting the rate distribution for various psychoses. A chapter on environment brings out the fact that mental disease in patients from urban areas is very much higher than in patients from rural areas. The chapter on marital status shows that the married population had much lower rates of mental disease than any of the other groups. Foreign-born persons showed the higher admission rate for mental disease (108 per 100,000) and the native-born a lower rate (91 per 100,000). In a chapter entitled, "Race and Mental Disease," the writer shows the age ratios and psychoses in Italians, Germans, Irish, English, Scandinavians and Negroes. Studying mental disease in relation to birth order, the author found no evidence that the first-born children are at a disadvantage so far as the appearance of a mental disorder is concerned. In studying economic status, it was found that unskilled workers appear to have the highest rate for mental disease. Illiteracy was also found to be related to high admission rates.

This volume makes a valuable addition to the literature of psychiatry and will probably be used as source material for some years to come. Oddly enough, its material parallels the data for Massachusetts published the same year by Dr. Neil A. Dayton.

Psychiatric Clinics for Children, with Special Reference to State Programs. By Helen Leland Witmer, Ph.D., Smith College School for Social Work. 8°, cloth, 437 pp. New York: The Commonwealth Fund, 1940. $2.50.

This book provides information for those interested in child-guidance clinic services. For the last nine years the National Committee for Mental Hygiene, through members of its field staff, has been conducting an evaluation of programs that offer this service throughout the country. The philosophy, the practice, the objectives and the achievements of clinical child psychiatry are studied. The results of this analysis, covering several years, are presented in this volume.

Part I describes the background of clinical child psychiatry in the United States. Part II discusses clinics financed by state governments, and evaluates typical policies. Part III suggests that the present difficulties of such clinics are traceable to lack of well-defined objectives and examines the alternative possibilities. The book ends with a list of basic requirements that, the author believes, must be met if a program of clinical psychiatry is to be successful.

The author is evidently thoroughly convinced of the superiority of the classic child-guidance clinic as developed in New York. The habit clinics, as developed in Massachusetts by Dr. Douglas A. Thom, and the traveling psychiatric school clinics, initiated by the late Dr. Walter E. Fernald, are discussed but briefly. The latter are described as "not wholly satisfactory." It seems that a traveling school clinic service which makes possible the examination of over 8000 children in about two hundred and seventy cities, towns and villages of Massachusetts each year should deserve serious consideration. At the end of 1940 the clinics mentioned had examined over 130,000 retarded and problem children in the public schools. The author gives as one of the reasons for the popularity of the Massachusetts school clinics that the recommendations advise the school authorities how "to get rid of the child."

Chapter 13, on psychiatric service for the feebleminded, is particularly weak and shows a lack of understanding of the subject. The author gives the impression that she is exasperated because the condition of mental deficiency cannot be explained by the Freudian philosophy.

There is a grave question in the minds of many whether or not the heavily staffed, cumbersome and expensive child-guidance clinic is a practical solution to present needs for psychiatric services for children. With thousands of children needing assistance, the existing child-guidance clinics serve hundreds. In essence this book is a plea for the continuation of the old order.

Principles of Hematology. By Russell L. Haden, M.D. Second edition, thoroughly revised. 8°, cloth, 362 pp., with 167 illustrations, 1 colored plate and 30 tables. Philadelphia: Lea and Febiger, 1940. $4.50.

The second edition of Haden's book has necessitated reprinting and revision within little more than a year. It is essentially a book for the general practitioner. Its chief features are its simple presentation, the large number of mechanistic diagrams of blood formation and blood destruction, and the illustrative cases. There are many excellent photomicrographs.

The text is unfortunately rather scanty, and there is frequent evidence of careless writing. In the interest of simplicity, thoroughness is frequently sacrificed, and occasionally a dogmatic attitude crops up. The volume index is still emphasized, although it is being more and more replaced by the corpuscular volume. There is no discussion of bone-marrow findings in various conditions, although it is certain that the intelligent practitioner would like to know about them. The anemias are discussed from illustrative cases only. The photoelectric-cell technic for the estimation of hemoglobin is not mentioned.

Despite these failings, a careful reading of the book by the practitioner should certainly advance his understanding of the field of hematology. The reviewer believes, however, that even the general practitioner should have the benefit of thoroughness in any subject he chooses to inquire into. This must be looked for in other books.

Haemorrhoids and Their Treatment: The varicose syndrome of the rectum. By Kasper Blond, M.D. Translated by E. Stanley Lee, M.S., F.R.C.S. 8°, cloth, 140 pp., with 49 illustrations. Baltimore: Williams and Wilkins Company, 1940. $4.50.

The author of this work has had an extensive experience with rectal and anal disease in Vienna and in clinics in other cities. His methods of treatment and his results are worthy of close study. He is an exponent of treating hemorrhoids, prolapse, fissure, pruritus and anal fistula by the injection method. The author believes all these conditions are due to one etiologic factor, — venous congestion and its complications, — an assumption that seems to be an oversimplification.

The operative treatment of anal fistula is deprecated because of the many failures; however, such poor results, in the opinion of the reviewer, are due not to the operation but to inadequate or improper postoperative care of the patient.

The methods and technics of the author are excellent, but few will agree wholly with his theories of the causation of anorectal disease.